FIRST AID FOR THE®

SURGERY clerkship

THIRD EDITION



- A survival manual for the surgery clerkship
- Tips on what you must know to excel on the shelf exam and impress on rounds
- Updated to reflect the latest protocols and guidelines

Latha Ganti • Matthew S. Kaufman
Nitin Mishra

Mc Graw Hill Education

FIRST AID FOR THE® Surgery Clerkship

Third Edition

Latha Ganti, MD, MS, MBa, FaCEP
Professor of Emergency Medicine and Neurology
University of Central Florida College of Medicine
Orlando, Florida

Matthew S. Kaufman, MD
Associate Director
Department of Emergency Medicine
Richmond University Medical Center
Staten Island, New York

nitin Mishra, MBBS, FaCS
Assistant Professor
Department of Surgery
Mayo Clinic
Phoenix, Arizona

New York Chicago San Francisco Athens London Madrid Mexico City
Milan New Delhi Singapore Sydney Toronto

First Aid for the® Surgery Clerkship, Third Edition

Copyright © 2017 by McGraw-Hill Education. All rights reserved. Printed in China. Except as permitted under the United States Copyright Act of 1976, no part of this publication may be reproduced or distributed in any form or by any means, or stored in a data base or retrieval system, without the prior written permission of the publisher.

Previous editions' copyright © 2009, 2003 by The McGraw-Hill Companies, Inc.

First Aid for the® is a registered trademark of McGraw-Hill Education Global Holdings, LLC.

1 2 3 4 5 6 7 8 9 DSS 21 20 19 18 17 16

ISBN 978-0-07-184209-9
MHID 0-07-184209-8

Notice

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The authors and the publisher of this work have checked with sources believed to be reliable in their efforts to provide information that is complete and generally in accord with the standards accepted at the time of publication. However, in view of the possibility of human error or changes in medical sciences, neither the authors nor the publisher nor any other party who has been involved in the preparation or publication of this work warrants that the information contained herein is in every respect accurate or complete, and they disclaim all responsibility for any errors or omissions or for the results obtained from use of the information contained in this work. Readers are encouraged to confirm the information contained herein with other sources. For example and in particular, readers are advised to check the product information sheet included in the package of each drug they plan to administer to be certain that the information contained in this work is accurate and that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in connection with new or infrequently used drugs.

This book was set in Electra LT Std by MPS Limited.
The editors were Catherine A. Johnson and Cindy Yoo.
The production supervisor was Catherine Saggese.
Project management was provided by Gaurav Prabhu.
The cover designer was Dreamit, Inc. Image: Farferros/shutterstock
RR Donnelley was printer and binder.

This book is printed on acid-free paper.

Library of Congress Cataloging-in-Publication Data

Names: Kaufman, Matthew S., author. | Ganti, Latha, author. | Mishra, Nitin, author.
Title: First aid for the surgery clerkship / Latha Ganti, Matthew S. Kaufman, Nitin Mishra.
Description: Third edition. | New York: McGraw-Hill Education, [2016] |
Matthew S. Kaufman's name appears first in the previous edition. |
Includes index.
Identifiers: LCCN 2016007057| ISBN 9780071842099 (pbk. : alk. paper) | ISBN 0071842098 (pbk. : alk. paper)
Subjects: | MESH: Surgical Procedures, Operative | Clinical Clerkship |
Examination Questions
Classification: LCC RD37.3 | NLM WO 18.2 | DDC 617.0076--dc23 LC record available at http://lccn.loc.gov/2016007057

McGraw-Hill Education books are available at special quantity discounts to use as premiums and sales promotions or for use in corporate training programs. To contact a representative, please visit the Contact Us pages at www.mhprofessional.com.

DeDicat ion

To my dad, Dr. Ganti L. Rao for constantly encouraging me to achieve; and to my children, Thor, Tej, Trilok, Karthik & Vaishnavi who put up with their mother's very busy career demands.

—Latha Ganti, MD, MS, MBA, FACEP

For Jenny G—New York's best

—Matthew S. Kaufman, MD

My work in this book is dedicated to my parents, Mr. Rajeshwar Mishra and Mrs. Indu Mishra.

—Nitin Mishra, MBBS, MS, MRCS (UK)

contents

Contributors vii
Faculty Reviewer viii
Introduction ix
How to Contribute xi

Section i: How to Succeed in the Surgery clerkship 1

Section ii: High-yield Facts for the Surgery clerkship 15

1 The Surgical Patient 17
2 Wounds 25
3 Acute Abdomen 35
4 Trauma 43
5 Critical Care 61
6 Fluids, Electrolytes, and Nutrition 73
7 The Esophagus 91
8 The Stomach 107
9 Small Bowel 123
10 Colon, Rectum, and Anal Canal 143
11 The Appendix 167
12 Hernia and Abdominal Wall Problems 175
13 The Hepatobiliary System 185
14 The Pancreas 215
15 The Endocrine System 229
16 The Spleen 257
17 The Breast 265
18 Burns 283

Section iii: High-yield Facts for Surgical electives 293

19 Anesthesia 295
20 Vascular Surgery 301
21 Pediatric Surgery 319
22 Ear, Nose, and Throat Surgery 335
23 Neurosurgery 361
24 Cardiothoracic Surgery 381
25 Transplant 401
26 The Genitourinary System 413
27 Orthopedics 429
28 The Hand 445

Section iv: classified: awards and opportunities 461

Index 477

contributors

awais ashfaq, Md
Resident, General Surgery
Mayo Clinic
Phoenix, Arizona
Cardiothoracic Surgery

Michael p. ast, Md
Director, Outpatient Joint Replacement
Mercer County Surgery Center
Director, Robotic Assisted Joint Replacement Program
Robert Wood Johnson University Hospital-Hamilton
Mercer Bucks Orthopaedics
Lawrenceville, New Jersey
Orthopedics
The Hand

ismael cubero, Md
Residency Site Director
Department of Emergency Medicine
New York City Health and Hospitals-Harlem
New York, New York
Trauma

onaona gurney, Md
Resident, Surgery
State University New York-Downstate Medical Center
Brooklyn, New York
Ward/Exam Tips

Michael J. klein, Md
Resident, Surgery
State University New York-Downstate Medical Center
Brooklyn, New York
Ward/Exam Tips

Nitin Mishra, MBBS, FacS
Assistant Professor
Department of Surgery
Mayo Clinic
Phoenix, Arizona
Acute Abdomen
Anesthesia
The Appendix
Burns
Colon, Rectum, and Anal Canal
Ear, Nose, and Throat Surgery
The Endocrine System
The Esophagus
Hernia and Abdominal Wall Problems
Neurosurgery
Pediatric Surgery
Small Bowel
The Stomach
The Surgical Patient
Wounds

Sneha Mishra, Md
Hospitalist
Overlook Medical Center
Summit, New Jersey
Fluids, Electrolytes, and Nutrition
The Spleen

kate X. peng, Md
Resident, General Surgery
General Surgery
Mayo Clinic
Phoenix, Arizona
Critical Care
The Breast
Vascular Surgery

david radvinsky, Md
Resident, General Surgery
State University New York-Downstate Medical Center
Brooklyn, New York
Ward/Exam Tips

vinay rai, Md, FacS
Assistant Professor
Department of Surgery
University of New Mexico
Albuquerque, New Mexico
Transplant

Sandeep a. Sathyanarayana, Md
Assistant Professor, Surgical Oncology
School of Medicine
Meharry Medical College
Nashville, Tennessee
Hepatobiliary System
Pancreas

denny Simon
Medical Student
Class of 2016
State University New York-Downstate College of Medicine
Brooklyn, New York
Ward/Exam Tips

Michael a. Zell, Md
Resident, Urology
Mayo Clinic
Phoenix, Arizona
Genitorurinary System

Faculty reviewer

deven unadkat, do
Associate Medical Director
Department of Emergency Medicine
Newark Beth Israel Medical Center
Newark, New Jersey

introduction

This book is designed in the tradition of the First Aid series of books. It is formatted in the same way as the other books in the series. You will f nd that, apart from preparing you for success on the clerkship exam, this resource will also help guide you in the clinical diagnosis and treatment of common surgical conditions.

The content of the book is based on the objectives for medical students as determined by the Association for Surgical Education (ASE). Each chapter contains the major topics central to the practice of general surgery and has been specif cally designed for the medical student. The book is divided into general surgery, which contains topics that comprise the core of the surgery rotation, and subspecialty surgery, which may be of interest but is generally considered less high yield for the clerkship. Knowledge of a subspecialty topic may be useful if observing a related surgery or if requesting a letter from a surgeon in that f eld.

The content of the text is organized in the format similar to other texts in the First Aid series. Topics are listed with bold headings, and the "body" of the topic provides essential information. The outside margins contain mnemonics, diagrams, summary or warning statements, and tips. Tips are categorized into Exam Tip, Ward Tip, and OR tip.

How to contribute

To continue to produce a high-yield review source for the surgery clerkship, you are invited to submit any suggestions or correction. Please send us your suggestions for:

- New facts, mnemonics, diagrams, and illustrations
- Low-yield facts to remove

For each entry incorporated into the next edition, you will receive personal acknowledgment. Diagrams, tables, partial entries, updates, corrections, and study hints are also appreciated, and significant contributions will be compensated at the discretion of the authors. Also let us know about material in this edition that you feel is low yield and should be deleted. You are also welcome to send general comments and feedback, although due to the volume of e-mails, we may not be able to respond to each of these.

The **preferred way** to submit entries, suggestions, or corrections is via **email.** Please include name, address, school affiliation, phone number, and e-mail address (if different from the address of origin). If there are multiple entries, please consolidate into a single e-mail or file attachment. Please send submissions to:

firstaidclerkships@gmail.com

Otherwise, please send entries, neatly written or typed to:

Latha G. Stead, MD
C/o Catherine A. Johnson
Executive Editor
McGraw-Hill Education
Two Penn Plaza, 9th Floor
New York, NY 10121

All entries become property of the authors and are subject to editing and reviewing. Please verify all data and spellings carefully. In the event that similar or duplicate entries are received, only the first entry received will be used. Include a reference to a standard textbook to facilitate verification of the fact. Please follow the style, punctuation, and format of this edition if possible.

note to contributors

All entries become properties of the authors and are subject to review and edits. Please verify all data and spelling carefully. In the event that similar or duplicate entries are received, only the first entry received will be used. Include a reference to a standard textbook to facilitate verification of the fact. Please follow the style, punctuation, and format of this edition if possible.

Section i

How to Succeed in the Surgery clerkship

In the Operating Room . . . 2

On the Wards . . . 3

- Be on Time 3
- Dress in a Professional manner 3
- Be Pleasant 3
- Be aware of the hierarchy 3
- address Patients and Staff in a respectful way 4
- Take responsibility for your Patients 4
- respect Patients' rights 4
- Volunteer 4
- Be a Team Player 4
- Be honest 4
- keep Patient information handy 5
- Present Patient information in an organized manner 5
- Presenting the chest radiograph (cxr) 5
- Types of notes 7
- Your Rotation Grade 8
- How to Study 9
- make a list of core material to learn 9
- as you see Patients, note Their major Symptoms and Diagnosis for review 10
- Select your Study material 10
- Prepare a Talk on a Topic 11
- Procedures 11

How to Prepare for the Clinical Clerkship Examination 11

- Study with friends 12
- Study in a Bright room 12
- eat light, Balanced meals 12
- Take Practice exams 12
- Tips for answering Questions 12

Pocket Cards for the Wards 13

The surgery clerkship is unique among all the medical school rotations. Even if you are dead sure you do not want to be a surgeon, it can be a very fun and rewarding experience if you approach it prepared. There are three key components to the rotation: (1) what to do in the OR, (2) what to do on the wards, and (3) how to study for the exam.

in the operating Room . . .

One of the most fun things on the surgery rotation is the opportunity to scrub in on surgical cases. The number and types of cases you will scrub in on depends on the number of residents and students on that service and how busy the service is that month. At some places, being able to go to the OR is considered a privilege rather than a routine part of the rotation. A few tips:

- **Eat before you begin the case.** Some cases can go on for longer than planned and it isn't cool to leave early because you are hungry (read unprepared!) or, worse, to pass out from exhaustion. As a student, your function in the OR will most likely be to hold retraction. This can be tedious, but it is important to pay attention and do a good job. Not pulling in the right direction obscures the view for your attending, and pulling too hard can destroy tissue. Many students get light-headed standing in one position for an extended period of time, especially when they are not used to it. Make sure you shift your weight and bend your knees once in a while so you don't faint. If you feel you are going to faint, then say something—ask one of the surgical techs to take over or state discreetly that you need relief. Do not hold on to the bitter end, pass out, and take the surgical field with you (believe it or not, this has actually happened; we print this advice from real experience).
- **Find out about the case as much as possible beforehand.** Usually, the OR schedule is posted the night before, so you should be able to tell. Read up on the procedure as well as the pathophysiology of the underlying condition. Know the important anatomic landmarks. Read up on the patient's H&P, with attention paid to PMH, PSH, medications, allergies, and relevant laboratory and radiology results.
- **Find out who you are working with.** If you can, do a quick bibliography search on the surgeon you will be working with. It can never hurt to know which papers (s)he has written, and this may help to spark conversation and distinguish you among the many other students they will have met.
- **Assess the mood in the OR.** The amount of conversation in the OR directed to you varies by attending. Some are very into teaching and will engage you during most of the surgery. Many others act as if you aren't even there. Some will interact if you make the first move; others nuke all efforts at interaction. You'll have to figure it out based on the situation. Generally, if your questions and comments reflect that you have read about the procedure and disease, things will go well.
- **Keep a log of all surgeries** you have attended, scrubbed on, or assisted with (see Table I-1). If you are planning to go into general surgery or a surgical subspecialty, it can be useful during residency interviews for conveying how much exposure/experience you have had. This is particularly true if your school's strength is clinical experience. The log can also be useful if you are requesting a letter from the chairman of surgery whom you have never worked with. It gives her/him an idea of what you have been doing with your rotation. Many rotations will set a minimum number of surgeries you are to attend. Try to attend as many as possible, and document them. This serves both to increase your exposure, and confirm your interest.

TABLE i-1. example of an operative case log

	Operation	S/O	attending	date	Mr #	Comments
1	cholecystectomy	S	Dr. Wolfe	5/28/2003	123456	Got to close, placed 28 nonabsorbable sutures
2	Appendectomy	S	Dr. tau	5/30/2003	246800	Used laparoscope
3	cataract surgery	o	Dr. Mia	6/1/2003	135791	

S, scrubbed in; O, observed; MR, patient's medical record number.

on the Wards . . .

Be on Time

Most surgical ward teams begin rounding between 6 and 7 a.m. If you are expected to "pre-round," you should give yourself at least 10 minutes per patient that you are following to see the patient and learn about the events that occurred overnight. Like all working professionals, you will face occasional obstacles to punctuality, but make sure this is occasional. When you first start a rotation, try to show up at least 15 minutes early until you get the routine figured out.

Dress in a Professional manner

Even if the resident wears scrubs and the attending wears stiletto heels, you must dress in a professional, conservative manner. Wear a short white coat over your clothes unless discouraged.

Men should wear long pants, with cuffs covering the ankle, a long collared shirt, and a tie. No jeans, no sneakers, no short-sleeved shirts.

Women should wear long pants or knee-length skirt, blouse, or dressy sweater. No jeans, no sneakers, no heels greater than 1½ inches, no open-toed shoes.

Both men and women may wear scrubs occasionally, especially during overnight call or in the operating room. Do not make this your uniform.

Be PleasanT

The surgical rotation is often difficult, stressful, and tiring. Smooth out your experience by being nice to be around. Smile a lot and learn everyone's name. If you do not understand or disagree with a treatment plan or diagnosis, do not "challenge." Instead, say "I'm sorry, I don't quite understand, could you please explain . . ." Be empathetic toward patients.

Be aware of The hierarchy

The way in which this will affect you will vary from hospital to hospital and team to team, but it is always present to some degree. In general, address your questions regarding ward functioning to interns or residents. Address your medical questions to attendings; make an effort to be somewhat informed on your subject prior to asking attendings medical questions.

Address Patients and Staff in a Respectful Way

Address patients as Sir, Ma'am, or Mr., Mrs., or Miss. Do not address patients as "honey," "sweetie," and the like. Although you may feel that these names are friendly, patients will think you have forgotten their name, that you are being inappropriately familiar, or both. Address attending physicians as "doctor" unless told otherwise.

Take Responsibility for your Patients

Know everything there is to know about your patients, their history, test results, details about their medical problem, and prognosis. Keep your intern or resident informed of new developments that he or she might not be aware of, and ask for any updates you might not be aware of. Assist the team in developing a plan and speaking to radiology, consultants, and family. Never give bad news to patients or family members without the assistance of your supervising resident or attending.

Respect Patients' Rights

1. All patients have the right to have their personal medical information kept private. This means do not discuss the patient's information with family members without the patient's consent, and do not discuss any patient in hallways, elevators, or cafeterias.
2. All patients have the right to refuse treatment. This means they can refuse treatment by a specific individual (you, the medical student) or of a specific type (no nasogastric tube). Patients can even refuse life-saving treatment. The only exceptions to this rule are if the patient is deemed to not have the capacity to make decisions or understand situations (in which case a health care proxy should be sought) and if the patient is suicidal or homicidal.
3. All patients should be informed of the right to seek advanced directives on admission. Often, this is done by the admissions staff, in a booklet. If your patient is chronically ill or has a life-threatening illness, address the subject of advanced directives with the assistance of your attending.

Volunteer

Be self-propelled, self-motivated. Volunteer to help with a procedure or a difficult task. Volunteer to give a 20-minute talk on a topic of your choice. Volunteer to take additional patients. Volunteer to stay late.

Be a Team Player

Help other medical students with their tasks; teach them information you have learned. Support your supervising intern or resident whenever possible. Never steal the spotlight, steal a procedure, or make a fellow medical student look bad.

Be Honest

If you don't understand, don't know, or didn't do it, make sure you always say that. Never say or document information that is false (a common example: "bowel sounds normal" when you did not listen).

keeP PaTienT inforMaTion handy

Use a clipboard, notebook, or index cards to keep patient information, including a miniature history and physical, lab, and test results at hand.

PresenT PaTienT inforMaTion in an organizeD manner

Here is a template for the "bullet" presentation:

> **This is a [age] year old [gender] with a history of [major history such as HTN, DM, coronary artery disease, CA, etc.] who presented on [date] with [major symptoms, such as cough, fever and chills], and was found to have [working diagnosis]. [Tests done] showed [results]. Yesterday the patient [state important changes, new plan, new tests, new medications]. This morning the patient feels [state the patient's words], and the physical exam is signif cant for [state major f ndings]. Plan is [state plan].**

The newly admitted patient generally deserves a longer presentation following the complete history and physical format.

Some patients have extensive histories. The whole history can and probably should be present in the admission note, but in ward presentation it is often too much to absorb. In these cases it will be very much appreciated by your team if you can generate a **good summary** that maintains an accurate picture of the patient. This usually takes some thought, but it's worth it.

PresenTing TheChesTraDiograPh (cXr)

3 "shuns" + "ABCDEFGHI"

1. Identification (identificay-SHUN)
   - correct patient, medical record number, date, and time.
2. Rotation (rotay-SHUN)
   - is this a posteroanterior (PA) or anteroposterior (AP) film?
   - are the clavicles at an equal height and can you see both medial convexities?
3. Penetration (penetray-SHUN)
   - the spinal column should become obscured halfway down the mediastinal silhouette.

A. **A**irway
   - trachea midline, without deviation
   - carina and bronchi visible, without distraction
   - look for presence and position of ETT—should be 2 cm above the carina
   - look for pneumomediastinum (perforation)

B. **B**ones
   - good inspiratory effort? (7–8 ribs visible)
   - look for rib/clavicular/humeral/sternal/scapular fractures or joint dislocations

C. **C**ostophrenic (angle)
   - look for blunting (hemothorax/pleural effusion)

D. **D**iaphragm
   - look for f attening (COPD), hemidiaphragm
   - look for free air under the diaphragm (pneumoperitoneum)

E. **E**sophagus
   - can't really see the esophagus, but can see presence of an NG or OG tube. Make sure that it's uncoiled, in the stomach, and not in the lung.
   - make sure the stomach is in the abdomen, not the chest (diaphragmatic hernia vs. rupture)
   - look for pneumomediastinum (perforation)

F. Lung **F**ields
   - look for airspace disease, opacities, pneumothorax, venous congestion, hilar lymphadenopathy
   - look for presence of chest tubes

G. **G**reat vessels
   - look for widening of mediastinum or mediastinal shift
   - look for presence of central venous catheters

H. **H**eart
   - look for cardiomegaly
   - look for retrocardiac opacities

I. **I**nterval
   - compare this f lm to the patient's previous f lms to see changes.

A sample CXR presentation may sound like:

> **This is the CXR of Mr. Jones. The f lm is an AP view with good inspiratory effort. There is an isolated fracture of the 8th rib on the right. There is no tracheal deviation or mediastinal shift. There is no pneumo- or hemothorax. The cardiac silhouette appears to be of normal size. The diaphragm and heart borders on both sides are clear; no inf ltrates are noted. There is a central venous catheter present, the tip of which is in the superior vena cava.**

The key elements of presenting a CXR are summarized in Table I-2.

TABLE I-2. How to Present a Chest Radiograph (CXR)

- First, confirm that the CXR belongs to your patient.
- If possible, compare to a previous film.

Then, present in a systematic manner:

1. Technique
   Rotation, anteroposterior (AP) or posteroanterior (PA), penetration, inspiratory effort.
2. Bony structures
   Look for rib, clavicle, scapula, and sternum fractures.
3. Airway
   Look for tracheal deviation, pneumothorax, pneumomediastinum.
4. Pleural space
   Look for f uid collections, which can represent hemothorax, chylothorax, pleural effusion.
5. Lung parenchyma
   Look for infiltrates and consolidations: These can represent pneumonia, pulmonary contusions, hematoma, or aspiration. The location of an infiltrate can provide a clue to the location of a pneumonia:
   - Obscured right (R) costophrenic angle = right lower lobe
   - Obscured left (L) costophrenic angle = left lower lobe

(continues)

TABLE I-2. How to Present a Chest Radiograph (CXR) (continued)

- Obscured Rheart border = right middle lobe
- Obscured Lheart border = left upper lobe

6. Mediastinum
- Look at size of mediastinum—a widened one (>8 cm) goes with aortic rupture.
- Look for enlarged cardiac silhouette (>1/2 thoracic width at base of heart), which may represent congestive heart failure (CHF), cardiomyopathy, hemopericardium, or pneumopericardium.

7. Diaphragm
- Look for free air under the diaphragm (suggests perforation).
- Look for stomach, bowel, or NGT tube above diaphragm (suggests diaphragmatic rupture).

8. Tubes and lines
- Identify all tubes and lines.
- An endotracheal tube should be 2 cm above the carina. A common mistake is right mainstem bronchus intubation.
- A chest tube (including the most proximal hole) should be in the pleural space (not in the lung parenchyma).
- An NGT tube should be in the stomach, and uncoiled.
- The tip of a central venous catheter (central line) should be in the superior vena cava (not in the right atrium).
- The tip of a Swan–Ganz catheter should be in the pulmonary artery.
- The tip of a transvenous pacemaker should be in the right atrium.

Types of NoTes

In addition to the admission H&P and the daily progress note, there are a few other types of notes you will write on the surgery clerkship. These include the preoperative, operative, postoperative, and procedure notes. Samples of these are depicted in Tables I-3 through I-6.

TABLE I-3. Sample Procedure Note (for Wound Repair)

Under sterile conditions following anesthesia with 5 cc of 2% lidocaine with epinephrine and negative wound exploration for foreign body, the laceration was closed with 3-0 Ethilon sutures. Wound edges were well approximated and no complications occurred. Wound was dressed with sterile gauze and triple antibiotic ointment.

TABLE I-4. Sample Preoperative Note

Pre-op diagnosis:	Abdominal pain
Procedure:	Exploratory laparotomy
Pre-op tests:	List results of labs (CBC, electrolytes, PT, aPTT, urinalysis), ECG, CXR (Most adult patients require coagulation studies; patients over 40 usually need ECG and CXR—these are institution specific.)
Blood:	How many units of what type were crossmatched and available; or, "none" if no blood needed
Orders:	For example, colon prep, NPO after midnight, preoperative antibiotics

TABLE i-5. Sample operative note

pre-op diagnosis:	Abdominal pain
post-op dx:	Small bowel obstruction
procedure:	Segmental small bowel resection with end-to-end anastomosis
Surgeon:	Dr. Attending
Assistant:	Your name Here
Anesthesia:	Get A (general endotracheal anesthesia)
	eBL (estimated blood loss): 100 cc
	Fluid replacement: 2000 cc crystalloid, 2 units FFP
	Uo = 250 cc
Findings:	10 cm of infarcted small bowel
	Dermoid tumor, left ovary
complications:	none

Wound was clean/clean contaminated/contaminated/dirty. (pick one)

closure: 0-0 prolene for fascia, 3-0 vicryl SQ staples for skin.

Procedure tolerated well, patient remained hemodynamically stable throughout. instrument, sponge, and needle counts were correct. Patient was extubated in the OR and transferred to the recovery room in stable condition.

TABLE i-6. Sample postoperative note

postoperative day:	1
procedure:	colon resection with diverting colostomy.
Vitals:	
intake and output:	For intake include all oral and parenteral fluids and tPn. For output include everything from all drains, tubes, and Foley.
physical examination:	note particularly lung and abdominal exam, and comment on wound site.
labs:	
Assessment:	
plan:	

Your Rotation Grade

Many students worry about their grade in this rotation. There is the perception that not getting honors in surgery pretty much closes the door to obtaining a residency spot in general or subspecialty surgery (ophthalmology, otorhinolaryngology, neurosurgery, plastic surgery, urology). While this is not necessarily true, the medicine and surgery clerkships are considered to be among the most important in medical school, so doing well in these is handy for all students. Usually, the clerkship grade is broken down into three or four components.

- Inpatient evaluation. This includes evaluation of your ward time by residents and attendings and is based on your performance on the ward. Usually, this makes up about half your grade and can be largely subjective.
- Ambulatory evaluation. This includes your performance in clinic, including clinic notes and any procedures performed in the outpatient setting.

- Written examination. Most schools use the NBME or "Shelf" examination. Some schools have their own homemade version, very similar to the NBME's. The test is multiple choice. This portion of the grade is anywhere from 20% to 40%, so performance on this multiple-choice test is vital to achieving honors in the clerkship. More on this below.
- Objective Structured Clinical Examination (OSCE). Some schools now include an OSCE as part of their clerkship evaluation. This is basically an exam that involves a standardized patient and allows assessment of a student's bedside manner and physical examination skills. This may comprise up to one-fourth of a student's grade. It is a tool that will probably become more and more popular over the next few years.

How to Study

Make a List of Core Material to Learn

This list should reflect common symptoms, illnesses, and areas in which you have particular interest, or in which you feel particularly weak. Do not try to learn every possible topic. The Association for Surgical Education (www.surgicaleducation.com) has put forth a manual of surgical objectives for the medical student surgery clerkship, on which this book is based. The ASE emphasizes:

Symptoms and Lab Tests

- Abdominal masses
- Abdominal pain
- Altered mental status
- Breast mass
- Jaundice
- Lung nodule
- Scrotal pain and swelling
- Thyroid mass
- Fluid, electrolyte, and acid–base disorders
- Multi-injured trauma patient

Common Surgeries

- Appendectomy
- Coronary artery bypass grafting (CABG)
- Cholecystectomy
- Exploratory laparotomy
- Breast surgery
- Herniorraphy
- Peptic ulcer disease (PUD) surgery
- Bariatric surgery

We also recommend:

- Preoperative care
- Postoperative care
- Wound infection
- Shock

The core of the general surgery rotation consists of the following chapters:

1. The Surgical Patient
2. Wounds

3. Acute Abdomen
4. Trauma
5. Critical Care
6. Fluids, Electrolytes, and Nutrition
7. The Esophagus
8. The Stomach
9. Small Bowel
10. Colon, Rectum, and Anal Canal
11. The Appendix
12. Hernia and Abdominal Wall Problems
13. The Hepatobiliary System
14. The Pancreas
15. Endocrine System
16. The Spleen
17. The Breast
18. Burns
20. Vascular Surgery
24. Cardiothoracic Surgery

The other chapters are somewhat less important, as they focus on subspecialty surgery. The subspecialty chapters are comprehensive and less "high yield" than the abdominal chapters, but they are an excellent primer for anyone considering going into subspecialty surgery. We kept the detail in these chapters due to feedback from several students who wanted a concise but comprehensive overview of surgical subspecialties.

You will notice that the chapters discuss pathophysiology and in general a lot of things that seem like they belong in a medicine book. The reason for this is that **the NBME clerkship exam covers the medicine behind surgical disease.** The exam does not ask specifics of operative technique. So, in a way, you are studying for three distinct purposes. The knowledge you need on the wards is the day-to-day management know-how. The knowledge you want in the OR involves surgical knowledge of anatomy and operative technique (see OR TIPs). The knowledge you want on the end of rotation examination is the epidemiology, risk factors, pathophysiology, diagnosis, and treatment of major diseases seen on a general surgery service.

as you see patients, note their major symptoms and diagnosis for review

Your reading on the symptom-based topics above should be done with a specific patient in mind. For example, if a patient comes to the office with a thyroid mass, read about Graves' disease, Hashimoto's, thyroid cancer, and the technique of needle aspiration in the review book that night.

select your study material

We recommend:

- This review book, First Aid for the Surgery Clerkship
- A major surgery textbook such as Schwartz's Principles of General Surgery (costs about $140), or Lawrence's Essentials of General Surgery.
- A full-text online journal database, such as www.mdconsult.com (subscription is $99/year for students)

- A small pocket reference book to look up lab values, clinical pathways, and the like, such as Maxwell Quick Medical Reference (ISBN 0964519119, costs $7)
- Free smartphone apps to look up drugs, such as Epocrates (Athenahealth, free)

Prepare a Talk on a Topic

You may be asked to give a small talk once or twice during your rotation. If not, you should volunteer! Feel free to choose a topic that is on your list; however, realize that this may be considered dull by the people who hear the lecture. The ideal topic is slightly uncommon but not rare, for example: bariatric surgery. To prepare a talk on a topic, read about it in a major textbook or a review article not more than 2 years old, and then search online or in the library for recent developments or changes in treatment.

Procedures

During the course of the surgery clerkship, there is a set of procedures you are expected to learn or at least observe. The common ones are:

- Intravenous line placement
- Nasogastric tube placement
- Venipuncture (blood draw)
- Foley (urinary) catheter placement
- Wound closure with sutures/staples
- Suture/staple removal
- Surgical knots (hand and instrument ties)
  – Ethicon Endosurgery (ethicon.com) provides free knot-tying training kits with instruction booklets for students. Simply go to their website, click "surgeons/clinicians" to contact a representative, and simply ask them to send one to your house.
- Dressing changes (wet to dry, saline, Vaseline gauze)
- Incision and drainage of abscesses
- Technique of needle aspiration (observe)
- Ankle–brachial index (ABI) measurement
- Evaluation of pulses with Doppler
- Skin biopsy (punch and excisional)
- Removal of surgical drains
- Transillumination of scrotum

How to Prepare for the clinical clerkship examination

If you have read about your core illnesses and core symptoms, you will know a great deal about the medicine of surgery. To study for the clerkship exam, we recommend:

2–3 weeks before exam: Read this entire review book, taking notes.
10 days before exam: Read the notes you took during the rotation on your core content list, and the corresponding review book sections.
5 days before exam: Read this entire review book, concentrating on lists and mnemonics.
2 days before exam: Exercise, eat well, skim the book, and go to bed early.
1 day before exam: Exercise, eat well, review your notes and the mnemonics, and go to bed on time. Do not have any caffeine after 2 p.m.

Other helpful studying strategies include:

Study with friends

Group studying can be very helpful. Other people may point out areas that you have not studied enough, and may help you focus on the goal. If you tend to get distracted by other people in the room, limit this to less than half of your study time.

Study in a Bright Room

Find the room in your house or in your library that has the best, brightest light. This will help prevent you from falling asleep. If you don't have a bright light, get a halogen desk lamp or a light that simulates sunlight (not a tanning lamp).

Eat Light, Balanced Meals

Make sure your meals are balanced, with lean protein, fruits and vegetables, and fiber. A high-sugar, high-carbohydrate meal will give you an initial burst of energy for 1–2 hours, but then you'll drop.

Take Practice Exams

The point of practice exams is not so much the content that is contained in the questions, but the training of sitting still for 3 hours and trying to pick the best answer for each and every question.

Tips for answering Questions

All questions are intended to have one best answer. When answering questions, follow these guidelines:

Read the actual question, then glance at the answers. For all questions longer than two sentences, reading the actual question stem (the last sentence) and then the answer choices first can help you sift through for key information.

"**Look for the words *EXCEPT, MOST, LEAST, NOT, BEST, WORST, TRUE, FALSE, CORRECT, INCORRECT, ALWAYS***, and NEVER**.** If you find one of these words, circle or underline it for later comparison with the answer.

Evaluate each answer as being either true or false. Example:

Which of the following is least likely to be associated with pulmonary embolism?

A. Tachycardia **T**
B. Tachypnea **T**
C. Chest pain? **F not always**
D. Deep venous thrombosis? **T not always**
E. Back pain **F? aortic dissection**

By comparing the question, noting LEAST, to the answers, "E" is the best answer.

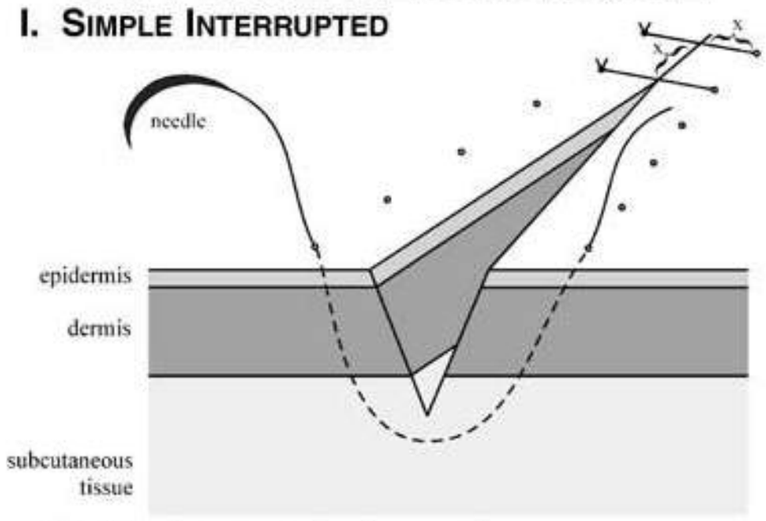
Finally, as the boy scouts say, "BE PREPARED."

Pocket cards for the Wards

The following "cards" contain information that is often helpful during the surgery rotation. We advise that you make a copy of these cards, cut them out, and carry them in your coat pocket when you are on the wards.

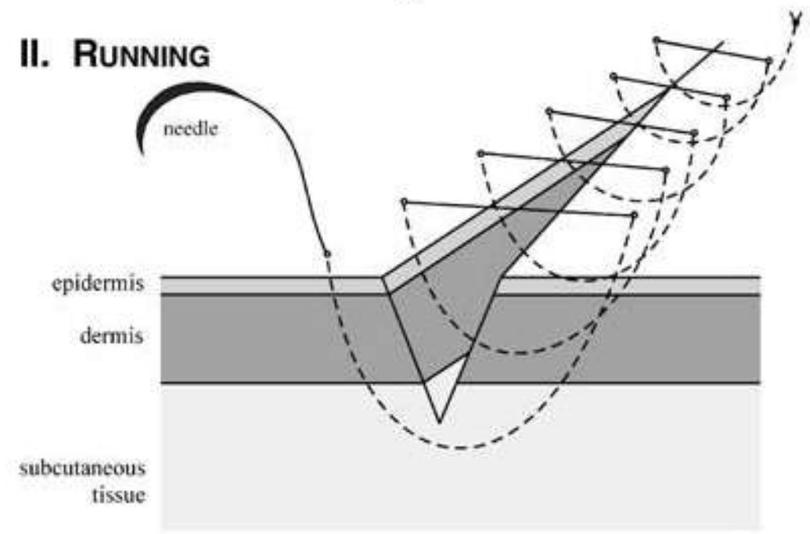
SUTURE TECHNIQUE

I. Simple Interrupted



- Used to close most simple wounds
- Edges should always be everted to prevent depression of scar. Do this by entering needle at 90 degrees to skin surface and follow curve of needle through skin.
- Entrance and exit point of needle should be equidistant from laceration.
- Do not place suture too shallow, as this will cause dead space.
- Use instrument tie or surgeons knot and place knot to one side of laceration not directly over laceration.

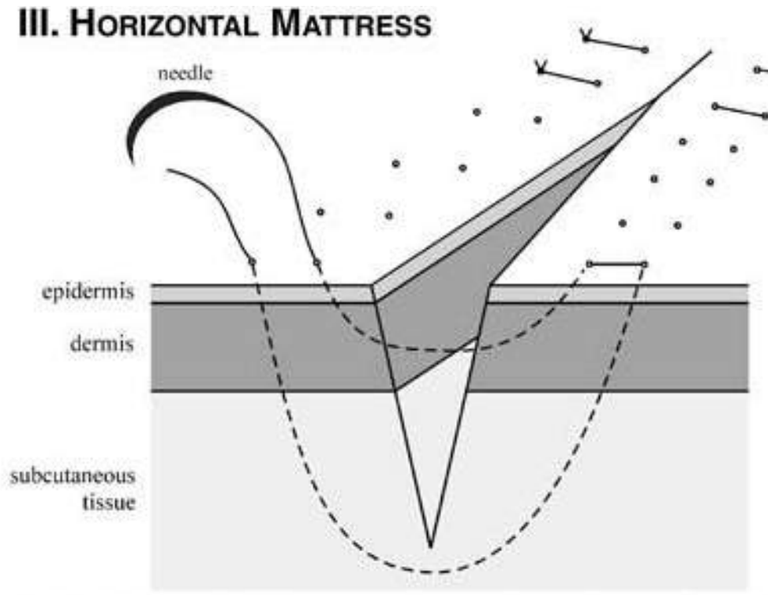
II. Running



- Not commonly used in the ED
- Disadvantage: One nicked stitch or knot means the entire suture is out.
- Advantage: Done well with sturdy knots, it provides even tension across wound.

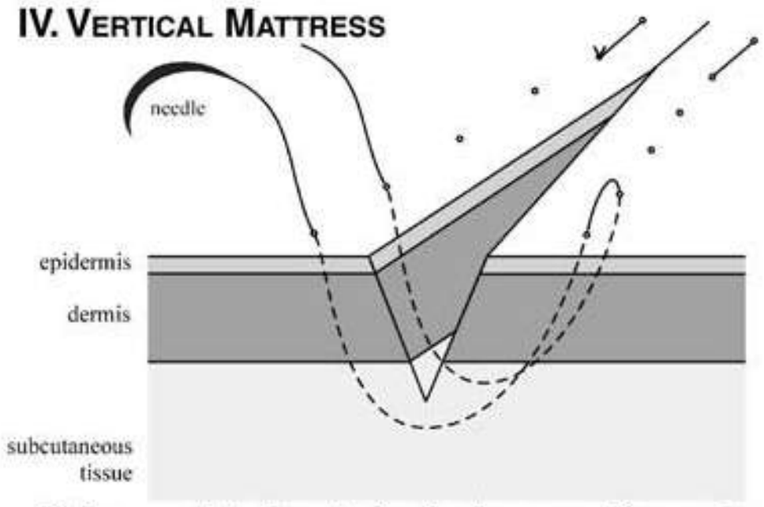
First Aid for the Surgery Clerkship, 2e; copyright © 2009 McGraw-Hill. All rights reserved.

III. Horizontal Mattress



- This suture also assists in wound edge eversion and helps to spread tension over a greater area.
- This stitch starts out like a simple interrupted suture; however, after the needle exits, it then enters again on the same side that it exited from only a few millimeters lateral to the stitch and equidistant from the wound edge and exits on the opposite side.

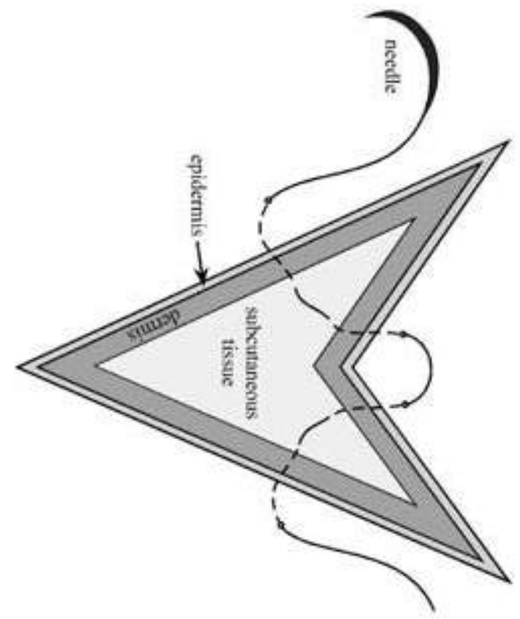
IV. Vertical Mattress



- This suture helps in reducing dead space and in eversion of wound edges.
- It does not significantly reduce tension on wound.
- The needle enters the skin farther away (more lateral) from the laceration than the simple interrupted and also exits further away on the opposite side.
- It then enters again on the same side that it just exited from but more proximal to the laceration and exits on the opposite side (where it originally entered) proximally.

First Aid for the Surgery Clerkship, 2e; copyright © 2009 McGraw-Hill. All rights reserved.

V. Corner Stitch



- This is used to repair stellate lacerations and help to preserve the blood supply to the tips of the skin.
- The needle enters the epidermis of the nonflap or nontip portion of the wound.
- It then enters the dermal layer of the skin tip on one side and proceeds through the dermal layer to exit the dermis on the other side of the tip (this portion will be buried).
- It then enters and exits the other side of the stellate wound. It will appear as a simple interrupted suture.

VI. Deep Sutures

- Absorbable
- Used for multilayered closure
- Deep sutures are absorbable because you will not be removing them.
- Use your forceps (pickups) to hold the skin from the inside of the wound.
- The first stitch is placed deep inside wound and exits superficially in dermal layer on same side of wound.
- Then it enters in the superficial dermal layer on the opposite side and exits deep.
- Tie a square knot and cut the tail of the suture close to the knot, which is called a buried knot.
- Now proceed with your superficial closure of the skin with nonabsorbable sutures.

First Aid for the Surgery Clerkship, 2e; copyright © 2009 McGraw-Hill. All rights reserved.

First Aid for the Surgery Clerkship, 2e; copyright © 2009 McGraw-Hill. All rights reserved.

Location	Suture size & type	Suture Technique	Removal
Scalp	3-0 or 4-0 nylon or polypropylene	Interrupted in galea, single tight layer in scalp, horizontal mattress if bleeding not well controlled	7–12 days
Pinna	5-0 Vicryl/Dexon in perichondrium	Close perichondrium with interrupted Vicryl and dose skin with interrupted nylon	3–5 days
Eyebrow	4-0 or 5-0 Vicryl (SQ) 6-0 nylon for skin	Layered closure	3–5 days
Eyelid	6-0 nylon	Single-layer horizontal mattress or simple interrupted	3–5 days
Lip	4-0 Vicryl (mucosa) 5-0 Vicryl (SQ or muscle) 6-0 nylon (skin)	If wound through lip, close three layers (mucosa, muscle, skin); otherwise do two-layer closure	3–5 days
Oral cavity	4-0 Vicryl	Simple interrupted or horizontal mattress if muscularis of tongue involved	N/A
Face	6-0 nylon (skin) 5-0 Vicryl (SQ)	Simple interrupted for single layer, layered closure for full-thickness laceration	3–5 days
Trunk	4-0 Vicryl (SQ, fat) 4-0 or 5-0 nylon (skin)	Single or layered closure	7–12 days
Extremity	3-0 or 4-0 Vicryl (SQ, fat, muscle) 4-0 or 5-0 nylon (skim)	Single-layer interrupted or vertical mattress; apply splint if over a joint	10–14 days
Hands and feet	4-0 or 5-0 nylon	Single-layer closure with simple interrupted or horizontal mattress; apply splint if over a joint	7–12 days
Nail bed	5-0 Vicryl	Meticulous placement to obtain even edges, allow to dissolve	N/A

SECTION II

High-Yield Facts for the Surgery Clerkship

The Surgical Patient	17	Colon, Rectum, and Anal Canal	143
Wounds	25	The Appendix	167
Acute Abdomen	35	Hernia and Abdominal Wall Problems	175
Trauma	43	The Hepatobiliary System	185
Critical Care	61	The Pancreas	215
Fluids, Electrolytes, and Nutrition	73	The Endocrine System	229
The Esophagus	91	The Spleen	257
The Stomach	107	The Breast	265
Small Bowel	123	Burns	283

the surgical patient

Preoperative Evaluation 18
- Anesthetic history 18
- the ASA Physical Status Classification System 18
- evaluate airway 18

Cardiac Risk Assessment 18

Pulmonary Risk Assessment 18
- risk factors for Pulmonary complications 18
- Goals to reduce risks 19
- ways to decrease complications 19

Hepatic Risk Assessment 19
- cautions 20

Renal Risk Assessment 20
- Preoperative evaluation 20
- dialysis 20

Hematological Assessment 20
- Preoperative labs 20
- Anemia 20
- thrombocytopenia 20
- coagulopathy 21

Nutritional Assessment 21

Antibiotic Prophylaxis 21
- by type of surgery 21

General Postoperative Complications 21

Common Complications 22
- ileus 22
- Clostridium difficile colitis 22
- dvt/pe 22
- wound infection 23

Instructions to Patient 23
- npo 23
- bowel preparation 24
- usual medications 24

WARD TIP

Ask yourself: What does the patient need in order to undergo the operation with the lowest risk possible?

WARD TIP

Patients with aortic stenosis are at increased risk for ischemia, myocardial infarction (MI), and sudden death.

WARD TIP

Stress test is positive if ST depressions > 0.2 mV are present or if there is an inadequate response of heart rate to stress or hypotension.

EXAM TIP

- Goldman's Index: Top Risks
  - #1—CHF
  - #2—MI within 6 months
  - #3—Arrhythmia
  - #4—Age > 70
  - #5—Emergent Surgery

EXAM TIP

- Absolute Contraindication to Surgery: DKA

WARD TIP

Echo is concerning when there is evidence of aortic stenosis (AS) or if the ejection fraction (EF) is < 35%.

WARD TIP

Low-risk patients require only history and physical (H&P) and ECG. Higher-risk patients may require further workup as described above.

Preoperative Evaluation

- Thorough history and physical exam.
- Optimization of any medical problems (i.e., cardiac or pulmonary diseases).

Anesthetic history

Note any prior anesthetics and associated complications.

the ASA Physical Status classification system

See Anesthesia chapter.

evaluate airway

Mallampati Classification (Figure 1-1) predicts difficulty of intubation. Test is performed with the patient in the sitting position, the head held in a neutral position, the mouth wide open, and the tongue protruding to the maximum.

- Class I: Visualization of soft palate, fauces, uvula, anterior and posterior tonsillar pillars.
- Class II: Visualization of soft palate, fauces, uvula.
- Class III: Visualization of soft palate, base of uvula.
- Class IV: Nonvisualization of soft palate.

Cardiac Risk Assessment

- For a patient greater than 40 years old with no cardiac history: Obtain electrocardiogram (ECG); if normal, no further workup required.
- For a patient of any age with cardiac history, or for an older patient: Obtain ECG; consider stress test and echocardiogram.

Goldman's risk assessment for noncardiac surgery (see Figure 1-2).

Pulmonary Risk Assessment

risk factors for pulmonary complications

- Known pulmonary disease.
- Abnormal pulmonary function tests (PFTs) (FEV < 11, maximum breathing capacity < 50% predicted).
- Smoking.
- Age > 60.
- Obesity.

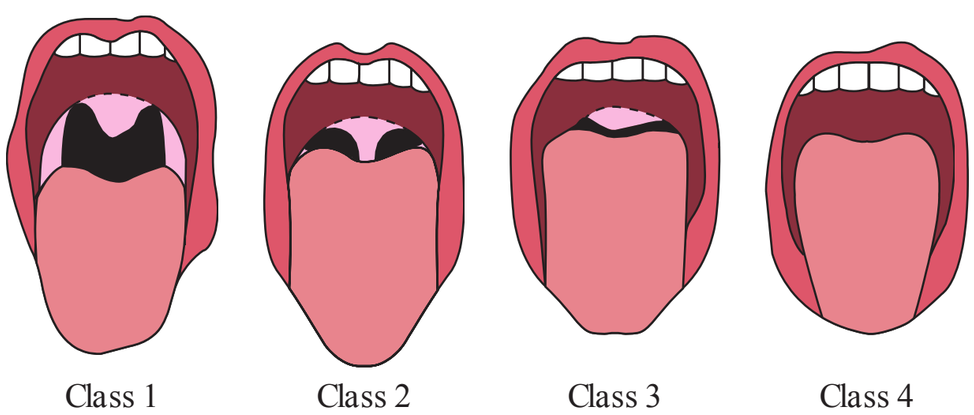


Figure 1-1. Mallampati classification of ease of intubation.

points		Probability of life-threatening complications
S3 gallop or JVD on exam	11	Add points to get risk 0–5 = class I = 1% 6–12 = class II = 5% 13–25 = class III = 11% >25 = class IV = 22%
MI within 6 months	10	
>5 PVCs/minute	7	
Rhythm other than sinus rhythm (SR) or SR with APCs on last ECG	7	
Age >70	5	
Emergent operation	4	
Intrathoracic, intraperitoneal, or aortic surgery	3	
Signif cant aortic stenosis	3	
Poor general medical condition	3	

Figure 1-2. goldman's risk assessment for noncardiac surgery. (Reproduced, with permission, from Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 297:845, 1977.)

- Upper abdominal or thoracic surgery.
- Long OR time.

Goals to reduce risks

- Decrease or cease smoking (benefit if at least 8 weeks preoperatively).
- Increase/optimize bronchodilator therapy.

ways to decrease complications

1. Incentive spirometry.
2. Early postop ambulation.
3. Chest physical therapy (PT).
4. Deep vein thrombosis (DVT) prophylaxis by sequential compression device (SCD) and subcutaneous heparin.

Hepatic Risk Assessment

There are two methods to determine Hepatic risk: The Child's Classification (see Table 1-1) and the Model for End-stage Liver Disease (MELD) (see below).

table 1-1. child's classif cation

Measure	1 point	2 points	3 points
Total bilirubin, µmol/L (mg/dL)	< 34 (< 2)	34–50 (2–3)	> 50 (> 3)
Serum albumin, g/dL	> 3.5	2.8–3.5	< 2.8
Prothrombin time, prolongation (seconds)	< 4.0	4.0–6.0	> 6.0
Ascites	None	Mild	Moderate to severe
Hepatic encephalopathy	None	Grade I–II (or suppressed with medication)	Grade III–IV (or refractory)

MELD Score

MELD = 3.78 × ln [serum bilirubin (mg/dL)] + 11.2 × ln [INR] + 9.57 × ln [serum creatinine (mg/dL)] + 6.43.

WARD TIP

Echocardiography:
- Sensitivity 90–100%
- Specificity 50–80%
- Fixed defects, or defects that persist with time, indicate infarcted or scarred tissue.
- Reversible defects are more concerning: Normal and fixed defects have similar negative predictive values for cardiac events.

EXAM TIP

Up to 35% of postoperative deaths are due to pulmonary complications.

WARD TIP

Major abdominal surgery decreases vital capacity by 50% and functional residual capacity by 30%.

WARD TIP

- FEV_1 < 70% predicted indicates increased risk.
- If VO_2 > 20, patient not likely to have pulmonary complications.

EXAM TIP

Heparin-induced thrombocytopenia (HIT).

WARD TIP

Because of the method by which they work (accentuate systemic thrombolysis) one SCD should work as well as two if one leg is injured.

WARD TIP

Atelectasis and/or pneumonia affect 20–40% of all postoperative patients.

ExAm TIP

Pneumonia has the highest morbidity and mortality of all pulmonary complications. The mortality of elderly patients with postoperative pneumonia is 50%.

WARD TIP

Remember that fuid mobilization typically occurs on postoperative day 2 or 3.

WARD TIP

NH_3 > 150: Mortality 80%
INR > 2: Mortality 40–60%

ExAm TIP

Increased BUN and creatinine indicate a loss of at least 75% of renal reserve. Intra- and postop hypotension must be strictly avoided in renal patients.

cAutions

- Watch for prolonged elevations in drug levels in patients with preoperative liver dysfunction.
- Acute hepatitis is relative contraindication to surgery.
- Attempt to control ascites prior to elective surgery, with fuid restriction, diuretics, and nutritional therapy.

Renal Risk Assessment

PreoPerAtive evAluAtion

- Check blood urea nitrogen (BUN) and creatinine.
- Estimate preoperative creatinine clearance (Cockcroft Gault equation):

$$[(140 - \text{age}) \times \text{Ideal body weight in kilograms}]/72 \times \text{Plasma creatinine (mg/dL)}$$

- Maintain intravascular volume.
- Ensure electrolytes are repleted; correct acidosis.
- Dialysis patients should be dialyzed within 24 hours prior to surgery to best control creatinine, electrolytes, and uremic platelet dysfunction.

diAlysis

- Overall mortality for dialysis-dependent patients: 5% (even when dialyzed within 24 hours of surgery).
- Acute renal failure that develops in perioperative period requiring dialysis is associated with a mortality of approximately 50–80%.
- Morbidity: Shunt thrombosis, pneumonia, wound infection, hemorrhage.

Hematological Assessment

PreoPerAtive lAbs

- Check complete blood count (CBC).
- Blood should be typed and crossed.

AnemiA

- Determine cause.
- Postpone elective operations.
- Patients with chronic hypoxia, ischemic heart disease, or cerebral ischemia do not tolerate anemia well.
- Sickle cell patients have increased risk of vaso-occlusive crises with operations. (This increased risk does not include patients with sickle cell trait.)

thrombocytoPeniA

See Table 1-2.

Table 1-2. Risk of Postoperative Bleeding by Platelet Count

Platelets	Likelihood of Bleed Perioperatively
> 150,000	Normal
100,000–150,000	Unlikely
50,000–100,000	Unlikely with adequate hemostasis
20,000–50,000	Possible excessive surgical bleeding
10,000–20,000	Spontaneous mucosal and cutaneous bleeding
< 10,000	Major spontaneous mucosal bleed, including GI tract

Coagulopathy

- Check prothrombin time (PT) and partial thromboplastin time (PTT) preoperatively.
- Note that elevated values should be expected in patients with liver disease.
- Factor abnormalities should be addressed (e.g., with hemophilia).

Nutritional Assessment

- Ideal body weight (IBW) = 50 kg + 2.3 kg/inch over 5 ft (male) or 45.5 kg + 2.3 kg/inch over 5 ft (female).
- Body mass index (BMI) = kg/m^2.
  - Loss of > 10% body weight in 6 months or a serum albumin level of < 3 is a poor prognostic indicator.

Antibiotic Prophylaxis

by Type of Surgery

1. In general: Cefazolin.
2. Gastrointestinal (GI) surgery: Cefazolin and metronidazole.
3. Urologic procedures: Ciprofoxacin.
4. Head and neck: Cefazolin or clindamycin and gentamicin.

General Postoperative Complications

- Immediate (0–24 hours):
  - Primary hemorrhage—either starting during surgery or following post-operative increase in blood pressure: Replace blood loss, and may require return to OR to reexplore.
  - Basal atelectasis.
  - Shock: Blood loss, acute MI, arrhythmias, pulmonary embolism (PE).
  - Oliguria: (hypotension intra- or postoperatively).

EXAM TIP

The risk of bleeding increases in patients with BUN > 100 due to platelet dysfunction, which can be corrected by desmopressin (DDAVP).

WARD TIP

The most common complication in dialysis is hyperkalemia (in nearly one third of patients).

WARD TIP

To determine source of a renal problem:

- FE_{Na} > 1 = intrinsic damage
- Specific gravity = 1.010 in ATN
- U_{Na} < 20 in prerenal

WARD TIP

- Risk of bleeding is further increased at any platelet level if patient is septic or has a functional platelet deficit.
- One unit of platelets raises platelet count by 5,000–10,000.

- Early (2 days–3 weeks):
  - Mental status changes due to electrolyte imbalance, dehydration, and sepsis.
  - Nausea and vomiting: Analgesia or anesthesia related; paralytic ileus.
  - Fever.
  - Secondary hemorrhage (infection).
  - Pneumonia.
  - Wound infection.
  - Wound or anastomotic dehiscence.
  - DVT, PE.
  - Acute urinary retention.
  - Urinary tract infection.
  - Postoperative wound infection.
  - Bowel obstruction due to fibrinous adhesions.
  - Paralytic ileus.
- Late (weeks to months):
  - Bowel obstruction due to fibrous adhesions.
  - Incisional hernia.
  - Nonhealing wound.
  - Recurrence of reason for surgery (e.g., malignancy).

WARD TIP

- Ideal BMI = 19–25.
- Increased risk if < 80% or > 12% of IBW or recent change of > 10% body weight.
- Consider baseline state of patient: Well-nourished with acute illness versus chronically ill, alcoholic, obese.

OR TIP

Antibiotic prophylaxis: Single dose 30 minutes prior to skin incision, and again 4 hours later if operation is ongoing.

WARD TIP

Return of bowel function: Small intestine, then stomach, then colon.

WARD TIP

To estimate when bowel function will return, allow one postoperative day per decade for major abdominal surgery.

Common Complications

ileus

- Incidence after GI surgery: 5%.
- Resolves with bowel rest and time in most patients.
- Check electrolytes! Hypokalemia can be a cause.

Clostridium difficile colitis

- Variable presentation, from mild diarrhea and discomfort to severe pain, tenderness, fever, and elevated white blood count (WBC).
- Associated with age, prior residence in nursing home, renal failure, immunocompromised state, antibiotic use, GI surgery, nasogastric tube (NGT) for > 48 hours.
- Treatment: Metronidazole or PO vancomycin.

1. Atelectasis
   - Most common cause of fever within 24 hours of surgery.
     - Reduced by early ambulation.
   - Physical exam shows reduced breath sounds in bilateral lung bases.
   - Treated by incentive spirometry and chest PT; no role of antibiotics.
2. Pneumonia
   - Characterized by fever, cough, dyspnea, pleuritic chest pain, purulent sputum.
   - Exam shows bronchial breath sounds, dullness, rales.
   - Chest x-ray shows infiltrates; sputum culture demonstrates causative organism.
   - Treated by organism-specific antibiotics.

dvt/Pe

- PE presents with tachycardia, tachypnea, and hypoxia when symptomatic (see Table 1-3).

TABLE 1-3. Well's criteria for PE

From history/exam	Score
DVT symptoms and signs (Homan's sign, calf enlargement)	3.0
PE is most likely diagnosis	3.0
Tachycardia (>100 bpm)	1.5
Recent surgery (in past 4 weeks) or immobilization for 3 or more days	1.5
Known history of confirmed PE or DVT	1.5
Hemoptysis	1.0
Treated for malignancy (current, within last 6 months, or under palliative care)	1.0

Score Interpretation

Total Score	Probability of PE	Relative Risk
Less than 2	3.6%	Low (recommend D-dimer to rule out)
2–6	20.5%	Intermediate
Greater than 6	66.7%	High (recommend CTA)

- Prevented by SCDs, subcutaneous heparin, and ambulation.
- PE diagnosed by ventilation/perfusion (V/Q) scan or chest computed tomography (CT) angiography.
- Treated by heparin infusion for segmental, sub-segmental PE. Massive PE is treated by surgery (embolectomy).

wound infection

See Wounds chapter.

Instructions to Patient

NPO

- To decrease the risk of aspiration with intubation, patients should refrain from solids 6–8 hours prior and from liquids 2–3 hours prior to surgery.
- In bowel surgery, when patients require bowel preps, the duration of NPO may be preceded by a day of clear liquids only with the prep to clear the bowel of stool and facilitate the operation.

WARD TIP

To simplify instructions, we tell patients to be NPO after midnight the night prior to elective surgery.

WARD TIP

Remember that the bowel prep is a source of iatrogenic fluid loss. Elderly or chronically ill patients may not tolerate this loss without IV fluid replacement.



WARD TIP

Aspirin and NSAIDs inhibit platelet activity and exacerbate bleeding. Holding Plavix in patients with drug-eluting cardiac stents may cause acute MI.

bowel PrePAr At ion

- Types:
  - Mechanical prep: Facilitates operation.
  - Oral antibiotics (neomycin, erythromycin base, metronidazole): Nadir bacterial count at commencement of operation if doses given at 1 p.m., 2 p.m., 11 p.m., the day prior (for a 7 a.m. case).

usuAl medicAt ions

- **Aspirin:** Avoid for 10 days preoperatively to allow platelets to regenerate.
- Hold clopidogrel (Plavix) for 7 days.
- **Warfarin (Coumadin):**
  - Three options: Avoid 3 days prior to operation and resume postoperative day 2; admit preoperatively and change to heparin, which can be held 2–4 hours ahead; operate through Coumadin.
- **Antihypertensives:** Continue, especially β-blockers; hold diuretics the morning of surgery.
- **Antithyroid medications:** Hold on morning of surgery.
- **Thyroid replacement:** Give on morning of surgery.
- **Oral hypoglycemics:** Avoid on day of surgery.
- **Insulin:** Give half usual dose on morning of surgery.



WARD TIP

$t_{1/2}$ thyroxine = 7 days so it can be held for several postoperative days without much effect.

Wounds

Introduction 26

Steps of Wound Healing 26
- Coagulation 26
- inflammation 26
- Collagen SyntheSiS 26
- angiogeneSiS (granulation) 26
- epithelialization 27
- ContraCtion and remodeling 27

Surgical Wound Classification 27
- Clean 27
- Clean-Contaminated 27
- Contaminated 27
- dirty/infeCted 27

Types of Wound Healing 28
- primary (firSt) intention 28
- SeCond intention 28
- third (delayed primary) intention 29

Factors Affecting Wound Healing 29

Wound Infections 29
- ClaSSifiCation 29
- pathophySiology 30

Common Surgical Pathogens 30
- SignS and SymptomS of Wound infeCtionS 31

Antimicrobial Prophylaxis 31
- general prinCipleS 31

Other Wound Complications 32
- hematoma 32
- Seroma 32
- Wound failure (dehiSCenCe and inCiSional hernia) 32
- CompliCationS of exCeSS SCar formation 33

Introduction

By definition, the end result of any surgical case is wound healing. Surgeons define a successful surgical case as one in which the patient survives, the pathology is removed and/or corrected, and the patient's wound heals. To accomplish this, it is important to understand the processes involved in wound repair, and ways in which these processes can lead to complications.

EXAM TIP

Phases of wound healing:
Hemostasis and Inflammation
Proliferation
Maturation
Remodeling

Steps of Wound Healing

- Coagulation
- Inflammation
- Collagen synthesis
- Angiogenesis
- Epithelialization
- Contraction

Coagulation

- Begins following wound formation.
- Coagulation and complement cascades are activated, and platelets create a hemostatic plug.
- Release of various inflammatory mediators from activated platelets sets the stage for the steps that follow.
- Impaired by anticoagulants, antiplatelet agents, and coagulation factor deficiency. Also may see impaired coagulation in the setting of chronic liver disease and uremia.

Inflammation

- The signs of inflammation are pain, swelling, heat, erythema, and loss of function.
- Inflammation occurs as a result of the wound's being invaded by polymorphonuclear neutrophils (PMNs, from the initial wound through the first 48 hours), and macrophages (peak numbers at 24 hours).
- Macrophages are essential for wound healing.
- Bacteria, cellular debris, and other foreign materials are also cleared from the wound site by the macrophages and PMNs.
- Impaired by steroids and other immunosuppressants, congenital, or acquired immune-deficient states.

EXAM TIP

Macrophages are essential for wound healing.

Collagen Synthesis

- Occurs by fibroblasts in the vicinity of the wound, in response to various growth factor peptides.
- The amino acids hydroxyproline and hydroxylysine are components of collagen. Their synthesis and hydroxylation is dependent on Fe, α-ketoglutarate, and ascorbate (vitamin C).

WARD TIP

The presence of granulation tissue is reassuring evidence that the healing process is under way.
At 1 month postinjury, the wound is at approximately 85% of its ultimate cohesive strength.

Angiogenesis (granulation)

- Occurs in response to peptide growth factors such as vascular endothelial growth factor (VEGF).
- Presence of these new vascular networks is what gives granulation tissue its characteristic beefy-red appearance.

epithelialization

- Occurs with the migration of epithelial cells over the wound defect.
- Integrity of the basement membrane is restored as type IV collagen and other matrix components are deposited.
- Foreign bodies, such as suture material, and necrotic tissue remain separated from the wound by the migrating epithelial cells.
- Once this step has occurred, the wound is essentially waterproofed.

Contraction and remodeling

- Process by which the surrounding uninjured skin is pulled over the wound defect and the size of the scar is reduced.
- Made possible by the action of myofibroblasts, which possess a contraction mechanism similar to that seen in muscle cells.
- A long process that takes many months before it is complete.
- Do not confuse wound contraction with scar contracture, as the latter occurs after wound repair has ceased. Scar contracture can lead to undesirable effects since architecture of the surrounding tissue may become distorted.
- Maturation of the scar occurs over the next 9 months to 2 years, characterized by cross-linking of collagen and clinical flattening of the scar.

Surgical Wound Classification

Clean

For a wound to be considered "clean," the following must be true:

- Wound created in a sterile and nontraumatic fashion, in an area that is free of preexisting inflammation.
- The respiratory, alimentary, genital, or urinary tract was not entered.
- All persons involved in the case maintained strict aseptic technique.

Clean-Contaminated

- The respiratory, alimentary, genital, or urinary tract was entered, but there was no significant spillage of its contents (e.g., feces), and there was no established local infection.
- There was only a minor break in aseptic technique.

Contaminated

- There was gross spillage from the gastrointestinal tract during the procedure.
- The genitourinary and biliary tracts were entered in the presence of local infection (e.g., cholangitis).
- The wound was the result of recent trauma.
- There was a major break in aseptic technique.

dirty/infected

- The wound was the result of remote trauma and contains devitalized tissue and/or purulent material.
- There is established infection or perforated viscera prior to the procedure.

WARD TIP

Epithelialization of surgical wounds closed primarily is usually complete by 24–48 hours.

EXAM TIP

Platelets are the first cells to act in wound healing, followed by macrophages and neutrophils. Fibroblasts appear last.

WARD TIP

Risk of infection for wounds:
Clean—2%
Clean-contaminated—5%
Contaminated—15%
Dirty—35%

WARD TIP

Generally, clean traumatic lacerations are closed with sutures or staples (primary intention) if less than 6–8 hours old.

Types of Wound Healing

Primary (First) Intention

See Figure 2-1.

- Type of healing seen following closure of clean surgical wounds, or traumatic lacerations in which there is minimal devitalized tissue, and minimal contamination.
- Edges of the incisional defect are approximated with the use of sutures or staples.
- Since the defect is very small, reepithelialization occurs rapidly, and overall healing time is short.
- Wounds closed primarily may have their dressing changed after 24–48 hours. By this time, epithelialization should be complete.
- Wound strength reaches 70–80% that of normal skin at 3 months.

Second Intention

- Type of healing seen following closure of wounds that are not approximated with sutures.
- Reason for not using sutures may be (1) that the wound edges cannot be apposed because the defect is very large (e.g., donor site of skin graft) or (2) that the surgeon chooses not to close the wound primarily because of the high risk of infection.

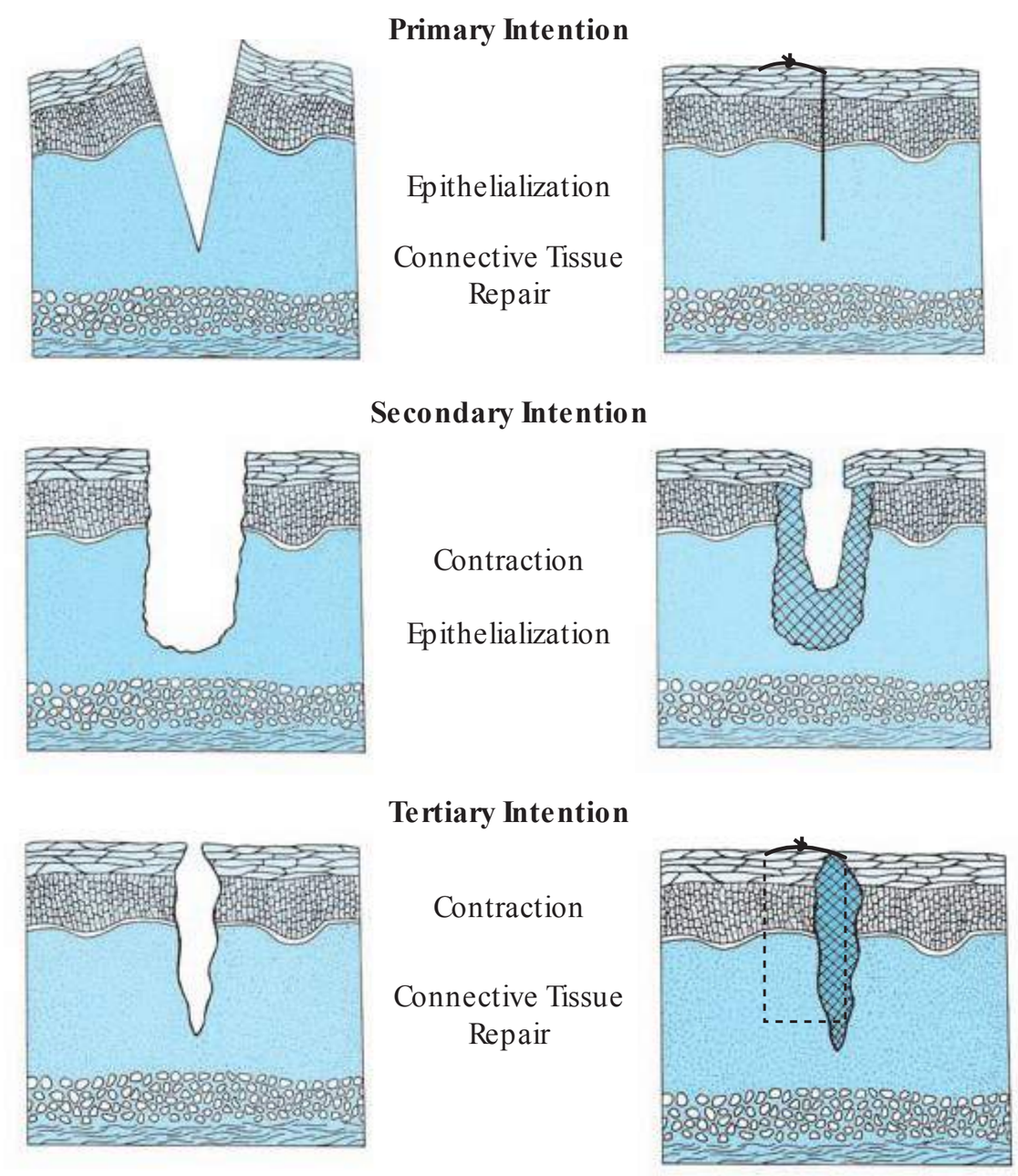


FIGURE 2-1. different clinical approaches to the closure and healing of acute wounds.
(Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 234.)

TABLE 2-1. Factors Affecting Wound Healing

Systemic	Local
Age	Mechanical injury
Nutrition	Infection
Trauma	Edema
Metabolic diseases	Ischemia/necrotic tissue
Immunosuppression	Topical agents
Connective tissue disorders	Ionizing radiation
Smoking	Low oxygen tension
	Foreign bodies

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 235.

- Wounds healing by second intention should be packed loosely with moist gauze and covered with a sterile dressing. The wound should be assessed daily for the development of granulation tissue and the presence of infection.
- Wound closes by contraction and then epithelialization. Contraction occurs from the edges of the wound inward, via contraction of myofibroblasts. Epithelialization then proceeds at 1 mm per day, also from the edges inward. Granulation tissue is generated at the center of the closing wound.

third (delayed primary) intention

- Type of healing seen following closure of wounds in which there is obvious gross contamination at the incisional site (i.e., the wound is classified as contaminated or dirty).
- An example of where delayed primary closure is often used is following removal of a ruptured appendix. In such cases, the parietal peritoneum and fascial layers are closed, and antibiotics are administered. The skin and subcutaneous tissue are not sutured until 3–5 days later after bacterial contamination has decreased.

Factors Affecting Wound Healing

See Table 2-1.

Wound Infections

Classification

Surgical site infections (SSIs) generally occur within 30 postoperative days. Depending on their location, they may be classified as superficial incisional, deep incisional, or organ/space infections.

- **Superficial incisional SSIs** exist when there is involvement by infection of the skin and subcutaneous tissue in the vicinity of the incision.
- **Deep incisional SSIs** exist when there is involvement by infection of deeper soft tissues, such as fascia or muscle, that were divided by the incision.

WARD TIP

Sutures are utilized in primary and delayed primary intention healing only.

WARD TIP

"Don't take down a dressing until after POD #2 unless told to!"

WARD TIP

Sutures are tied to approximate, not strangulate, a healing wound!

- **Organ/space SSIs** exist when there is infection of any anatomical structure remote from the incisional site but manipulated during the procedure.

Early SSIs that occur in the first 24 hours postoperatively are most commonly due to Streptococcus or Clostridium. These bacteria grow very fast because they excrete enzymes that digest local tissue and impair host defenses. Infections due to other bacteria generally become apparent later (4–5 days postoperatively) because they lack such virulence factors.

WARD TIP

Infections that involve both deep and superficial tissues are classified as deep incisional SSIs.

pathophySiology

Depends on:

- Factors relating to microorganism:
  - The critical number is 10^5 (dose of contaminating microorganisms required to define it as a wound infection).
- Factors relating to the patient—increased risk with:
  - Infection that is remote from the surgical site.
  - Diabetes.
  - Smokers.
  - Immunosuppressive agents, such as corticosteroids.
  - Severe protein–calorie malnutrition.
  - AIDS, disseminated malignancy, and any other immunocompromised state.
- Factors relating to surgical technique:
  - Preoperative considerations:
    - Patient skin preparation and the administration of antimicrobial prophylaxis.
  - Intraoperative considerations:
    - Strict attention to aseptic technique.
    - OR should be well ventilated and maintained under positive pressure to prevent the entrance of pathogens from the corridor.
  - Postoperative considerations:
    - Proper wound management and discharge instructions are key.
    - Wound management will differ depending on whether the wound is closed by primary, secondary, or delayed primary intention (see above).

WARD TIP

Many of the factors that impair wound healing will also increase the risk of wound infections.

WARD TIP

Hair in the vicinity of the incision should be trimmed immediately before the procedure with clippers.

WARD TIP

In the healthy individual, the lower respiratory tract and the upper urinary tract are sterile.

Common Surgical Pathogens

- The organism responsible for causing an SSI is best identified by culturing the involved region.
- The most likely causative organism can be predicted based on the site of the operation:
  - ***Staphylococcus aureus* and coagulase-negative staphylococci** are commonly isolated from wounds that follow thoracic (cardiac and noncardiac), neurological, breast, ophthalmic, vascular, and orthopedic surgery.
  - **Gram-negative bacilli and anaerobes** are commonly isolated from wound infections that develop following appendectomy, colorectal, biliary tract, OB/GYN, and urological cases.
  - **Streptococci and oropharyngeal anaerobes** are commonly isolated from wounds that follow head and neck procedures in which the mucosa of the oral cavity is involved.
  - Animal bite infections: Pasteurella multocida; also Streptococcus and Staphylococcus.

Signs and Symptoms of Wound Infections

- Classic signs:
  - Calor (heat, warmth)
  - Rubor (redness)
  - Tumor (swelling)
  - Dolor (pain)
- More severe infections may produce systemic symptoms such as fever, chills, and rigors.
- Unusual for a wound infection to cause fever before postoperative day 3.
- Other causes of postoperative fever include urinary tract infection (UTI) (owing to prolonged placement of a Foley catheter), deep vein thrombosis (DVT)/thrombophlebitis, and certain medications.

Diagnostic Approach

- Physical exam should be directed at the surgical wound, looking for signs of infection such as induration, warmth, erythema, or frank purulent discharge.
- Any discharge present should be sent for microbiological culture.
- The febrile patient should be evaluated with an appropriate fever workup: complete blood count (CBC), blood and urine cultures, urinalysis, and chest x-ray should be sent.
- If an intra-abdominal abscess is suspected, a computed tomography (CT) scan of abdomen should be done.
- Lumbar puncture may be required for the patient with fever and altered mental status, especially in a patient that is status post craniotomy for a neurosurgical procedure.

Treatment

- Wound abscesses (superficial SSIs) require incision and drainage followed by thorough irrigation.
- Deeper SSIs may require surgical debridement.
- Systemic antibiotic therapy is required for deep SSIs; they may or may not be required for superficial SSIs, depending on the severity of the infection.
- Peritoneal abscesses (organ/space SSI) may be treated by CT-guided percutaneous drainage; those that cannot be drained percutaneously require open drainage.

EXAM TIP

The 5 W's of postoperative fever:
- Wound infection
- Wind (atelectasis)—most common cause of postop fever within 1 day after surgery (caused by IL-1 release from alveolar macrophages)
- Water (UTI)
- Walking (DVT)
- Wonder drugs (direct side effects or those compromising immunity, respiration, or perfusion)

EXAM TIP

Atelectasis is the most common cause of postoperative fever within the first 24 hours.

EXAM TIP

- "Salmon-colored" drainage from a postop abdominal wound—think fascial dehiscence.

Antimicrobial Prophylaxis

The benefits of surgical antimicrobial prophylaxis are maximized if the chosen antibiotic:

- Provides appropriate coverage against the most probable contaminating organisms.
- Is present in optimal concentrations in serum and tissues at the time of incision.
- Is maintained at therapeutic levels throughout the operation.

General Principles

- For gram-positive cocci: First- and second-generation cephalosporins.
- For gram-negative rods: Third-generation cephalosporins.

- For anaerobes: Metronidazole or clindamycin.
- For gram-negative rods: Aminoglycosides.
- For methicillin-resistant Staphylococcus aureus: Vancomycin.
- All choices of antibiotics should be tailored based on the resistance profile of the community your hospital serves.

Other Wound Complications

hematoma

- Collection of blood that may form in the vicinity of a surgical wound.
- Small hematomas may be left alone and allowed to reabsorb spontaneously, while larger hematomas may require drainage.

Seroma

- Collection of fluid in the vicinity of a wound that is not blood or pus.
- Due to creation of a potential space combined with disruption of local draining lymphatic channels (e.g., mastectomy).
- Small seromas may be left alone and allowed to reabsorb spontaneously, while larger ones may require aspiration.

Wound failure (dehiscence and incisional hernia)

definitions

- Occurs when there has been complete or partial disruption of one or more layers of the incisional site.
- Termed **dehiscence** if it occurs early in the postoperative course before all stages of wound healing have occurred (complete disruption).
- Termed **incisional hernia** when it occurs months or years after the surgical procedure (at least the skin is intact, i.e., partial disruption).

Causes

Poor operative techniques that may lead to wound failure include the following:

- Suture material with inadequate tensile strength. Since absorbable sutures lose their tensile strength rather quickly, nonabsorbable sutures should be used to close the fascia.
- Inadequate number of sutures. Sutures should be placed no greater than 1 cm apart; if placed greater than 1 cm apart, herniation of viscera may occur between sutures.
- Too small bite size. Sutures should be placed no less than 1 cm from the wound edge; if placed closer to the wound edge, the fascia may tear.
- Stitches tied too tight (ischemia).

Patient factors may be divided into:

1. Systemic illnesses that impair wound healing, such as malnutrition, corticosteroid therapy, sepsis, uremia, liver failure, or poorly controlled diabetes.
2. Physical factors that place stress on the incisional site, such as coughing/retching, obesity, and the presence of ascites.

Treatment

- Immediate treatment of wound dehiscence involves minimizing contamination of the operative site by the placement of sterile packing. The patient must then be brought back to the OR to reclose the incision.
- Incisional hernia must be treated promptly, especially if the patient is symptomatic (e.g., abdominal pain, nausea, vomiting). This is because strangulation of the bowel may occur, resulting in necrosis and increased morbidity. Incisional hernias are repaired by repairing the fascial defect, with or without the use of a synthetic mesh to reinforce the defect.

Complications of excess scar formation

Hypertrophic scar and keloid formation (both are raised above skin level):

- Keloids spread beyond the margins of the original wound and are painful.
- Common in African-Americans (genetic predisposition).
- Commonly seen around the earlobes and the deltoid, presternal, and upper back regions.
- Hypertrophic scars usually subside spontaneously, whereas keloids need treatment with intralesional corticosteroid injection, topical application of silicone sheets, or the use of radiation or pressure. Surgery is reserved for excision of large lesions or as second-line therapy when other modalities have failed.

Acute Abdomen

Acute Abdomen 36
- Definition 36
- History 36

Physical Exam 37

Diagnosis 37

Management 38

Surgical Causes of Abdominal Pain 38
- right Upper QUaDrant (r UQ) 38
- right Lower QUaDrant (r LQ) 39
- Left Lower QUaDrant (LLQ) 39
- Left Upper QUaDrant (LUQ) 39
- DiffUse 39
- periUmbiLicaL 41
- SUprapUbic 41

Important Nonsurgical Causes of Abdominal Pain 41

Acute Abdomen

Definition

Abrupt onset of abdominal pain usually accompanied by one or more peritoneal signs (i.e., rigidity, tenderness (with or without rebound), involuntary guarding). Most causes of acute abdomen are surgical.

History

Pain is the most common presenting feature of an acute abdomen. Special attention to the characteristics of the pain will aid in reaching the diagnosis.

Location

Visceral pain: Poorly localized, usually dull, achy pain arising from distention or spasm in hollow organs. Example: Crampy pain *f*elt during early intestinal obstruction.

Mid-epigastrium: Stomach, duodenum, hepatobiliary system, pancreas.
Mid-abdomen: Jejunum, ileum.
Lower abdomen: Colon, internal reproductive organs.

Parietal pain: Sharp, well-localized, somatic pain arising from irritation (usually by pus, bile, urine, or gastrointestinal secretions) of the parietal peritoneum. Example: Inf amed appendix causing sharp right lower quadrant (RLQ) pain due to irritation *of* nearby peritoneum.

Quality of Pain

Steady pain is most common, but differentiating character of pain is helpful:

Gradual, steady pain
Acute cholecystitis
Acute cholangitis
Hepatic abscess
Diverticulitis

Intermittent, colicky pain
Small bowel obstruction
Inflammatory bowel disease
Biliary colic

Abrupt, excruciating pain
Perforated ulcer
Ruptured aneurysm
Ureteral colic

Rapid-onset, severe constant pain
Acute pancreatitis
Ectopic pregnancy
Mesenteric ischemia
Strangulated bowel
Acute appendicitis

Precipitating or Palliative Factors

May include:

- Change in position.
- Association with food (better, worse).
- Pain that wakes one from sleep (signif cant).

Radiation

- Biliary tract pain may radiate to the right shoulder or right scapula (due to right hemidiaphragmatic irritation).
- Splenic rupture pain may radiate to left shoulder.
- Kidney pain may radiate from flank to groin and genitalia (loin to groin).
- Pancreas pain may radiate to back.

WARD TIP

Assessment of pain should include PQRST:
Precipitating or palliative factors
Quality of pain: Stabbing, shooting, boring, dull
Radiation
Severity
Timing

EXAM TIP

- Think mesenteric ischemia in acute abdomens with blood in stool (positive guaiac).
- Think ruptured AAA in acute abdomen with back pain and/or blood in urine (communicating fistula).

WARD TIP

Kehr's sign is pain referred to the left shoulder due to irritation of the left hemidiaphragm. Often seen with splenic rupture and residual pneumoperitoneum after laparoscopy.

Associated Symptoms

- Vomiting: Pain relieved by vomiting is supportive of small bowel obstruction (SBO), afferent loop syndrome.
- Bilious vomiting is a clue for proximal SBO.
- Bowel habits: Constipation or obstipation (no stool or flatus) is suggestive of bowel obstruction.
- Mucoid diarrhea with blood (red currant jelly stool) is seen in intussusception.
- Anorexia: Very nonspecific symptom; however, most patients with acute appendicitis will have anorexia.
- Fever: Seen in appendicitis, acute cholecystitis, diverticulitis, acute pancreatitis, pyelonephritis.

medical and Surgical History

- Past abdominal surgery? Any abdominal surgery increases the chance of SBO secondary to adhesions (even years later).
- Atrial fibrillation: Increased risk for mesenteric ischemia (embolism) because of emboli to mesenteric arteries.
- Menstrual and sexual history (acute salpingitis vs. pelvic inflammatory disease vs. ruptured ectopic).

Physical Exam

Things to look for:

- Vital signs: Most patients with a surgical abdomen will have vital sign abnormalities secondary to pain, inflammation, fluid and electrolyte derangements, and anxiety.
- General: Hydration status, mentation, nutritional status.
- Chest: Auscultation.
- Abdomen: See Tables 3-1 and 3-2.

Diagnosis

Initial laboratory evaluation should include:

- CBC.
- Electrolytes.
- Amylase, lipase.

TABLE 3-1. Steps in Physical examination of the Acute Abdomen

1. Inspection	7. Punch tenderness
2. Auscultation	Costal area
3. Cough tenderness	Costovertebral area
4. Percussion	8. Special signs
5. Guarding or rigidity	9. External hernias and male genitalia
6. Palpation	10. Rectal and pelvic examination
One finger	
Rebound tenderness	
Deep	

Reproduced, with permission, from Doherty GM. Current Surgical Diagnosis & Treatment. 12th ed. New York: McGraw Hill; 2006: 484.

WARD TIP

Serial abdominal exams and observation may be necessary in cases in which the etiology of abdominal pain is initially unclear.

WARD TIP

Pain of perforated ulcer is severe and of sudden onset.
Murphy's sign is seen in cholecystitis.

WARD TIP

Contrast for abdominal CT:
For the most optimal imaging, both oral and IV contrast is used. In some cases (such as impaired renal function or allergy to IV contrast) this is not feasible. Noncontrast CT, although suboptimal for most cases (except nephrolithiasis), still provides lots of information.

tAbLe 3-2. Physical Findings in Various causes of Acute Abdomen

Condition	Helpful SignS
Perforated viscus	Scaphoid, tense abdomen; diminished bowel sounds (late); loss of liver dullness; guarding or rigidity.
Peritonitis	Motionless; absent bowel sounds (late); cough and rebound tenderness; guarding or rigidity.
Inflamed mass or abscess	Tender mass (abdominal, rectal, or pelvic); punch tenderness; special signs (Murphy's, psoas, or obturator).
Intestinal obstruction	Distention; visible peristalsis (late); hyperperistalsis (early) or quiet abdomen (late); dif use pain without rebound tenderness; hernia or rectal mass (some).
Paralytic ileus	Distention; minimal bowel sounds; no localized tenderness.
Ischemic or strangulated bowel	Not distended (until late); bowel sounds variable; severe pain but little tenderness; rectal bleeding (some).
Bleeding	Pallor, shock; distention; pulsatile (aneurysm) or tender (e.g., ectopic pregnancy) mass; rectal bleeding (some).

Reproduced, with permission, from Doherty GM. Current Surgical Diagnosis & Treatment. 12th ed. New York: McGraw-Hill; 2006: 484.

- Electrocardiogram (ECG) to rule out myocardial infarction (MI) and also as a preoperative baseline cardiac assessment.
- Liver function tests (LFTs) for right upper quadrant (RUQ) pain.
- β-hCG (human chorionic gonadotropin) for all women of childbearing age.
- Chest x-ray (CXR) and abdominal x-ray (AXR) to look for free air (can detect as little as 1–2 mL); easier to see under right hemidiaphragm. Presence of stomach bubble obscures it on the left (see Figure 3-1).
- Abdominal computed tomography (CT) should be used after the above assessment is complete and the diagnosis remains elusive (e.g., young male with clinical signs and symptoms of appendicitis should not undergo CT) (see Figure 3-2).

WARD TIP

The pain of appendicitis localizes to McBurney's point.
Physical exam signs in appendicitis: Rovsing's, obturator, psoas (see Appendix chapter).

Management

- Early diagnosis improves outcome.
- Key is deciding whether surgical intervention is needed (see Table 3-3).

WARD TIP

Appendicitis is still the most common surgical emergency in the pregnant woman.

Surgical Causes of Abdominal Pain

rigHt Upper QUaDrant (rUQ)

Perforated duodenal ulcer
Acute cholecystitis
Hepatic abscess

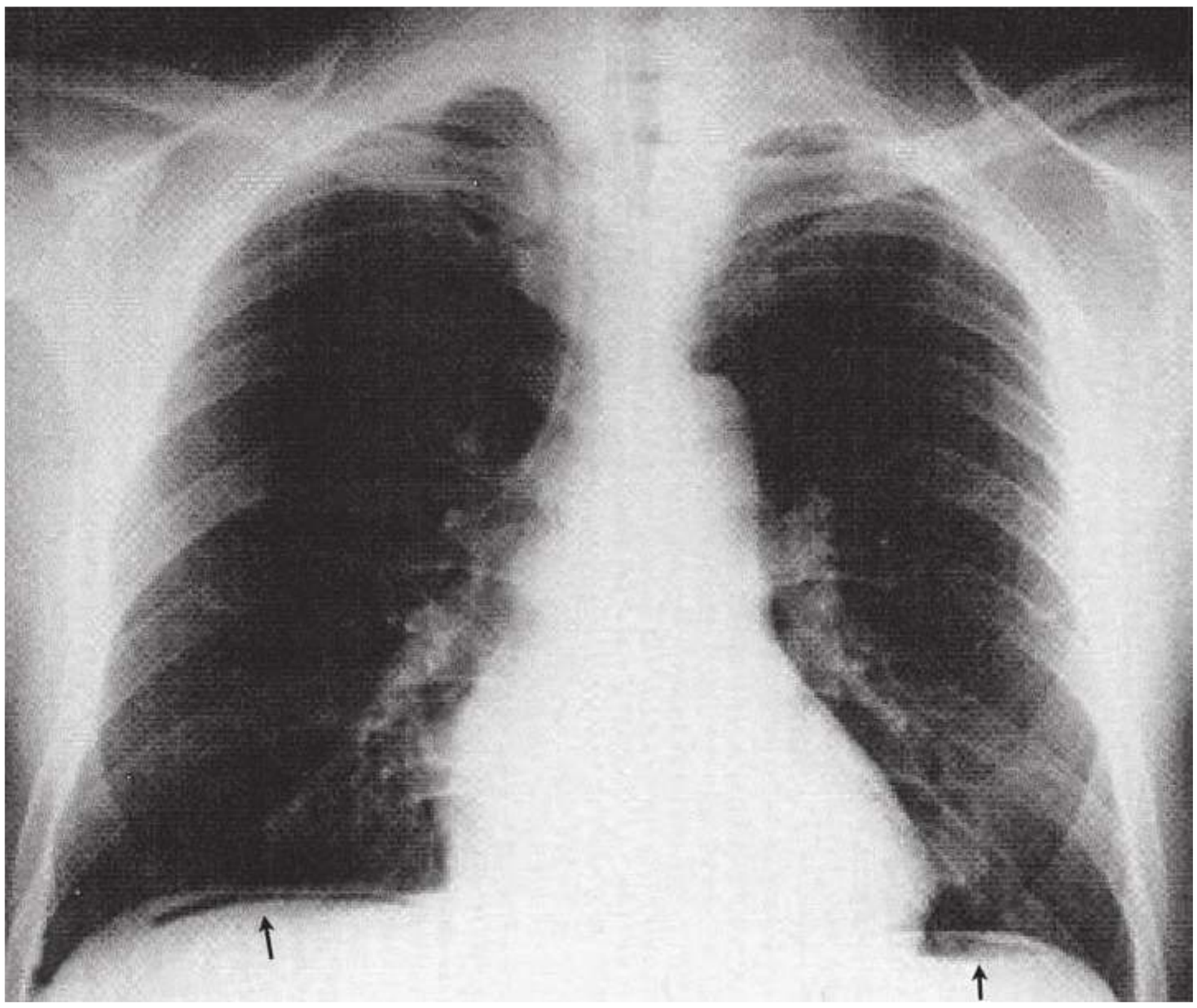


FIGURE 3-1. upright cXR demonstrating free air under both hemidiaphragms (arrows).
(Reproduced, with permission, from Billittier et al. Emerg Med Clin North Am. November 1996; 14(4):795.)

Retrocecal appendicitis
Appendicitis in a pregnant woman

right Lower QUadrant (rLQ)

Appendicitis
Cecal diverticulitis
Meckel's diverticulitis
Intussusception
Ovarian pathology (torsion/ectopic pregnancy/tubo-ovarian abscess)

Left Lower QUadrant (LLQ)

Sigmoid diverticulitis
Volvulus
Ovarian pathology (torsion/ectopic pregnancy/tubo-ovarian abscess)

Left Upper QUadrant (LUQ)

Splenic rupture
Splenic abscess

Diffuse

Bowel obstruction
Leaking aneurysm
Mesenteric ischemia

WARD TIP

Patients with splenic rupture will have an elevated white count in the setting of trauma.

WARD TIP

Patients with bowel obstruction will initially be able to take in fluids by mouth, and vomit a short time afterward.

EXAM TIP

Abdominal aortic aneurysm (AAA): Pulsatile mass on physical exam.

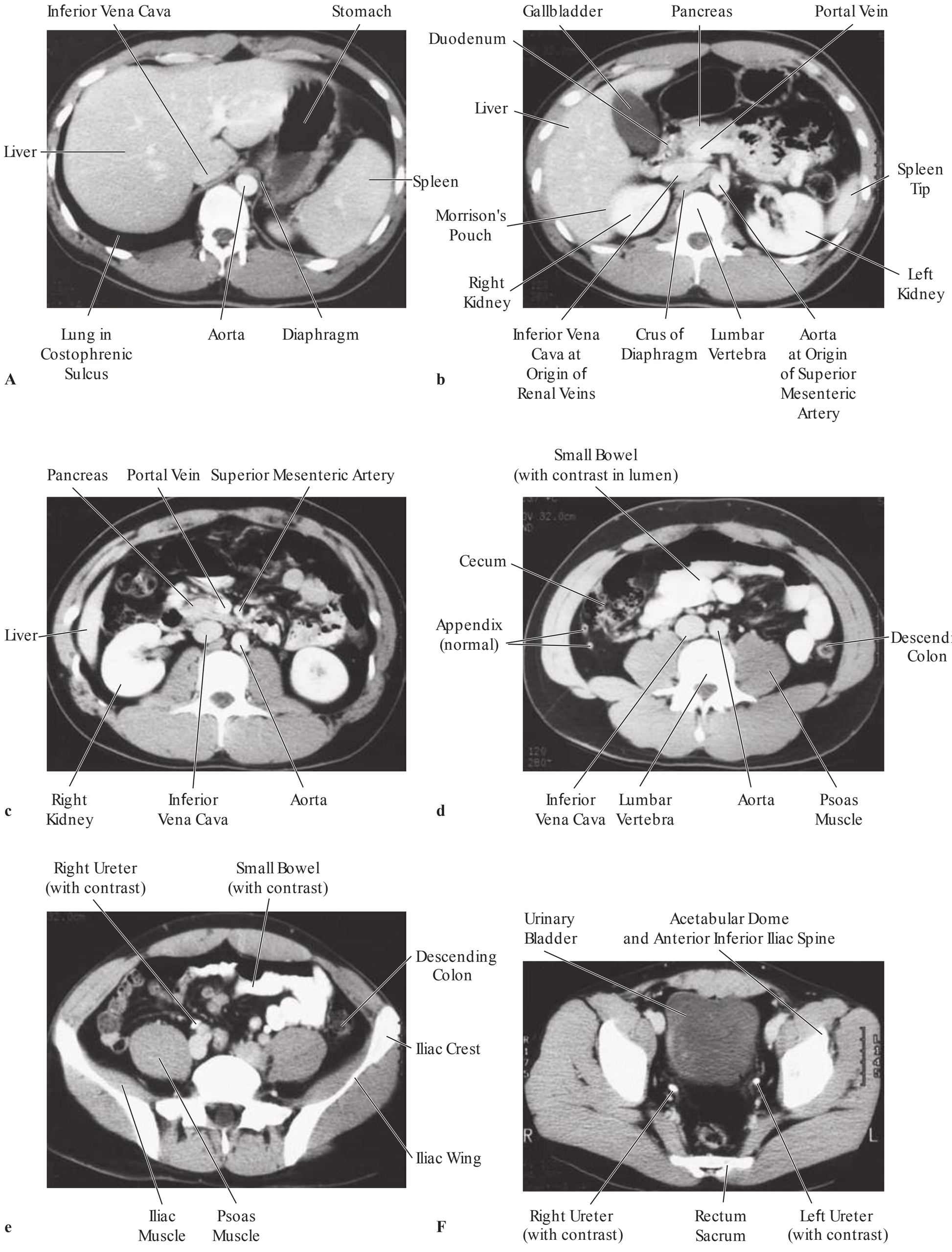


FIGURE 3-2. Abdominal CT anatomy. Normal abdominopelvic CT scan of a 26-year-old man. Both oral and intravenous contrast was administered. **A.** Liver (right and left lobes), stomach, and spleen. **B.** Liver, gallbladder, kidneys, and pancreas. **C.** Kidneys, pancreas, and intestines. **D.** Small bowel, cecum, ascending colon, and normal appendix. **E.** Intestines and ureters at level of iliac wings. **F.** Bladder, distal ureters, and rectum at the level of the acetabular domes. (Reproduced, with permission, from Schwartz DT, Reisdorf EJ, eds. Emergency Radiology. New York: McGraw-Hill; 2000: 519.)

TABLE 3-3. Indications for Urgent Operation in Patients with Acute Abdomen

Physical findings

- Involuntary guarding or rigidity, especially if spreading.
- Increasing or severe localized tenderness.
- Tense or progressive distention.
- Tender abdominal or rectal mass with high fever or hypotension.
- Rectal bleeding with shock or acidosis.
- Equivocal abdominal findings along with—
  - Septicemia (high fever, marked or rising leukocytosis, mental changes, or increasing glucose intolerance in a diabetic patient).
  - Bleeding (unexplained shock or acidosis, falling hematocrit).
  - Suspected ischemia (acidosis, fever, tachycardia).
  - Deterioration on conservative treatment.

Radiologic findings

- Pneumoperitoneum.
- Gross or progressive bowel distention.
- Free extravasation of contrast material.
- Space-occupying lesion on scan, with fever.
- Mesenteric occlusion on angiography.

Endoscopic findings

- Perforated or uncontrollably bleeding lesion.

Paracentesis findings

- Blood, bile, pus, bowel contents, or urine.

Reproduced, with permission, from Doherty GM. Current Surgical Diagnosis & Treatment. 12th ed. New York: McGraw-Hill; 2006: 491.

PERIUMBILICAL

Early appendicitis
Pain from SBO

SUPRAPUBIC

Ovarian pathology (torsion/ectopic pregnancy/tubo-ovarian abscess)
Psoas abscess
Incarcerated groin hernia
Sigmoid diverticulitis

Important Nonsurgical Causes of Abdominal Pain

Myocardial infarction
Mittelschmerz
Poisoning (lead, black widow spider)
Herpes zoster
Lower lobe (RLL) pneumonia
Endocrine (Addisonian crisis, diabetic ketoacidosis)
Sickle cell crisis
Hypercalcemia
Porphyrias
Psychological (hysteria)

WARD TIP

Fitz–Hugh–Curtis syndrome is perihepatitis associated with chlamydial infection of cervix.

WARD TIP

MI: Do an ECG on all patients presenting with midepigastric pain.

WARD TIP

Pain of pancreatitis is described as boring radiating straight to the back.

Trauma

Advanced Trauma Life Support (ATLS) 44
- "Golden Hour" of Trauma 44
- Primary Survey 44
- ABCs 44

Resuscitation 45
- Intravenous fluid 45
- Shock 45
- Radiologic and diagnostic studies 45
- Secondary Survey 46
- Tetanus Prophylaxis 46

Head Trauma 46

Neck Trauma 46
- General 46
- anatomy 46
- Penetrating injuries 46
- resuscitation 47
- management 47

Spinal Trauma 48

Thoracic Trauma 48
- Pericardial Tamponade 48
- Blunt Cardiac Trauma 48
- Pneumothorax 48
- Tension Pneumothorax 49
- Hemothorax 49

Abdominal Trauma 50
- General 50
- Physical examination 50
- focused abdominal sonography for Trauma (FAST) 51
- diagnostic peritoneal lavage (DPL) 53
- CT Scanning 54
- angiography 54
- Serial Hematocrits 54
- laparoscopy 54
- indications for exploratory laparotomy 54
- Types of injury: General approach 54
- liver injury 54
- Splenic Trauma 55
- Bowel injury 55

Pancreatic Injury 57
- General 57
- diagnosis 57
- Treatment 57

Genitourinary (GU) Trauma 57
- General 57
- Signs and Symptoms 57
- urinalysis 58
- retrograde urethrogram 58
- retrograde cystogram 58
- Bladder rupture 58
- ureteral injury 58
- renal injury 58

Advanced Trauma Life Support (ATLS)

"Golden Hour" of Trauma

Period immediately following trauma in which rapid assessment, diagnosis, and stabilization must occur.

Primary Survey

Initial assessment and resuscitation of vital functions. Prioritization based on ABCs of trauma care.

aBCs

- **A**irway (with cervical spine precautions)
- **B**reathing and ventilation
- **C**irculation (and **C**ontrol of hemorrhage)
- **D**isability (neurologic status)
- **E**xposure/**E**nvironment control
- **F**oley/**F**ingerstick/**F**AST exam

airway and C-spine

- Assess patency of airway.
- Use jaw thrust or chin lift initially to open airway.
- Clear foreign bodies or suction airway if necessary.
- Insert oral or nasal airway when needed.
- Obtunded/unconscious patients should be intubated (GCS < 8).
- Surgical airway = cricothyroidotomy—used when unable to intubate.
- Expanding hematoma, emphysema in the neck, noisy breathing, and soot around nares (inhalation injury) all require considering intubation.

Breathing and Ventilation

- Inspect, auscultate, and palpate the chest.
- Ensure adequate ventilation and identify and treat injuries that may immediately impair ventilation:
  - Tension pneumothorax
  - Flail chest and pulmonary contusion
  - Massive hemothorax
  - Open pneumothorax

Control of Hemorrhage

- Place two large-bore peripheral (14- or 16-gauge) IVs.
- Consider intraosseous line placement or central line sheath introducers in patients with difficult access.
- Assess circulatory status (capillary refill, pulse, skin color) (see Shock section).
- Control life-threatening hemorrhage using direct pressure.

Disability

- Rapid neurologic exam.
- Establish pupillary size and reactivity and level of consciousness using the AVPU or Glasgow Coma Scale.

WARD TIP

ALWAYS follow your ABCs!

- The first step in management of a patient with a mangled, bleeding leg is to ASSESS THE AIRWAY. Don't get tunnel vision.

WARD TIP

Assume C-spine injury in trauma patients until proven otherwise.

WARD TIP

All trauma patients should receive supplemental O_2.

WARD TIP

Draw blood samples at the time of intravenous catheter placement.

EXAM TIP

AVPU scale:
Alert
Verbal
Pain
Unresponsive

Exposure/Environment/Extras

Completely undress all trauma patients.

Foley Catheter

- Placement of a urinary catheter is considered part of the resuscitative phase that takes place during the primary survey.
- Foley is contraindicated when urethral transection is suspected, such as in the case of a pelvic fracture. If transection suspected, perform retrograde urethrogram before Foley.

Signs of urethral Transection

- Blood at the meatus.
- A "high-riding" prostate.
- Perineal or scrotal hematoma.
- Be suspicious with any pelvic fracture.

Resuscitation

- Begins during the primary survey.
- Life-threatening injuries are tended to as they are identified.

inTravenouS fluid

- Fluid therapy should be initiated with 1 L of an isotonic (either lactated Ringer's or normal saline) crystalloid solution.
- Consider early use of blood and blood products in patients with signs of shock.
- Pediatric patients should receive an IV bolus of 20 cc/kg.
- After 2 boluses, patients that continue to be hypotensive should receive blood products until stabilized.

SHoCk

- Inadequate delivery of oxygen on the cellular level secondary to decreased end-organ perfusion.
- In traumatic situations, shock is the result of hemorrhage until proven otherwise.

See Critical Care chapter.

radioloGiCand diaGnoSTiCSTudieS: adjunCTS To Primary Survey

- X-rays of the chest and pelvis usually occur concurrently with early resuscitative efforts; however, this procedure should never interrupt the resuscitative process.
- Diagnostic peritoneal lavage (DPL) and focused abdominal sonogram for trauma (FAST) are also tools used for the rapid detection of intra-abdominal bleeding that often occurs early in the resuscitative process (see Abdominal Trauma).
- CT scans should be done only for patients who are hemodynamically stable.

WARD TIP

Don't forget to keep your patients warm (cover them up again as soon as possible).

WARD TIP

Examine prostate and genitalia before placing a Foley.

WARD TIP

Trauma resuscitation requires teamwork with many different activities overlapping in both time and space.

WARD TIP

The antecubital fossae are a good place to find nice veins in which to place large-bore IVs.

WARD TIP

Use warmed fluids whenever possible.

WARD TIP

Stages of shock with % of blood loss

- Think of it as a tennis match:
  - Stage 1: Up to 15%
  - Stage 2: Up to 30%
  - Stage 3: Up to 40%
  - Stage 4: > 40% (game over)

Secondary Survey

Trauma History

- Begins once the primary survey is complete and resuscitative efforts are well under way. Whenever possible take an **AMPLE** history:
  - **A**llergies
  - **M**edications/**M**echanism of injury
  - **P**ast medical history/**P**regnant?
  - **L**ast meal
  - **E**vents surrounding the mechanism of injury
- Head-to-toe evaluation of the trauma patient; frequent reassessment is key.
- Neurologic examination including the GCS (see Table 23-2), procedures, radiologic examination, and laboratory testing take place at this time if not already accomplished.

WARD TIP

"Fingers and tubes in every orifice."

Tetanus Prophylaxis

Immunize as needed.

EXAM TIP

The majority of the vital structures of the neck lie within the anterior triangle.

Head Trauma

See Neurosurgery chapter.

Neck Trauma

General

Described in broad terms as penetrating vs. blunt injuries even though considerable overlap exists between the management of the two.

Anatomy

- The neck is divided into **zones** (see Figure 4-1):
  - Zone I spans the area from the thoracic inlet to the cricoid cartilage.
  - Zone II lies between cricoid and the angle of the mandible.
  - Zone III lies above the angle of the mandible to the base of the skull.
- These divisions help drive the diagnostic and therapeutic management decisions for penetrating neck injuries.

WARD TIP

C-spine injuries are much more common with blunt neck injury.

Penetrating Injuries

Any injury to the neck in which the platysma is violated.

Vascular Injuries

- Very common and often life threatening.
- Can lead to exsanguination, hematoma formation with compromise of the airway, and cerebral vascular accidents (e.g., from transection of the carotid artery or air embolus).

WARD TIP

Fracture of the hyoid bone is suggestive of a significant mechanism of injury.

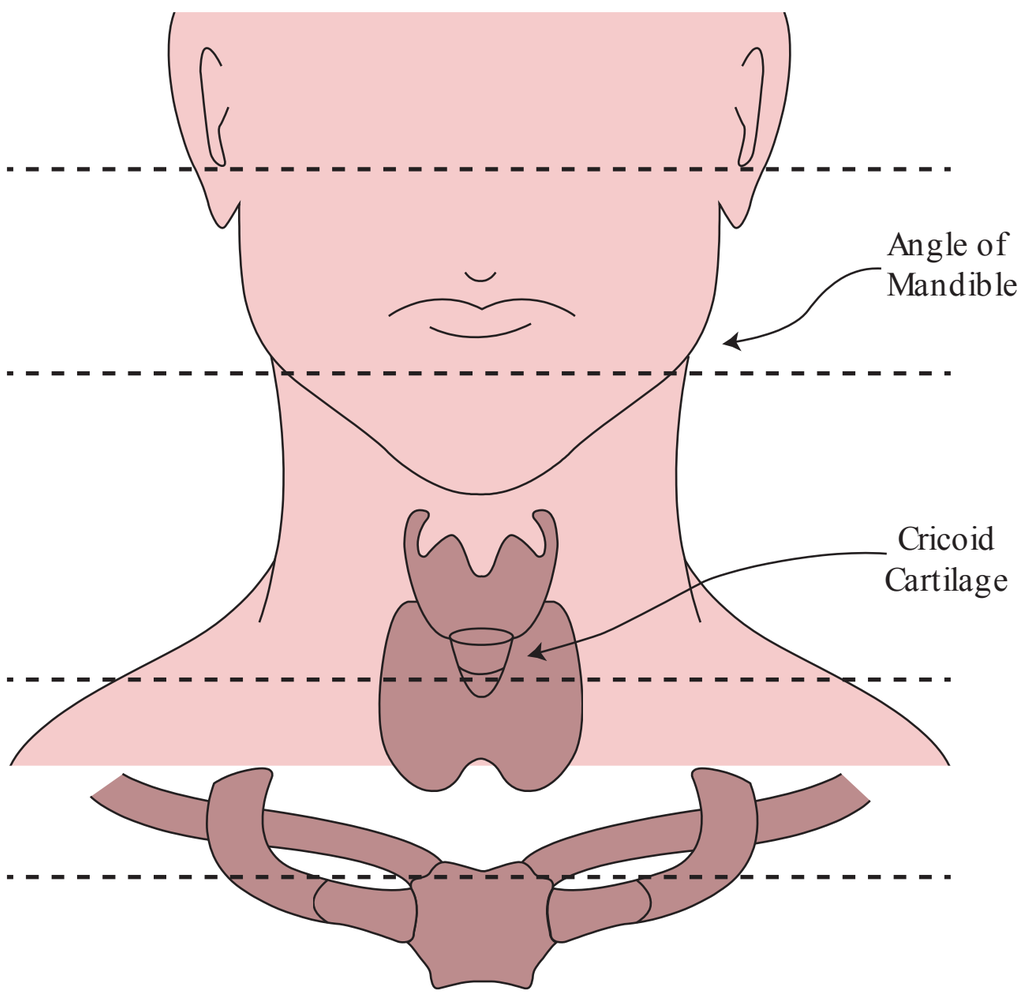


FIGURE 4-1. **Zones of the neck.**

Nonvascular Injuries

- Injury to the larynx and trachea including fracture of the thyroid cartilage, dislocation of the tracheal cartilages and arytenoids, for example, leading to airway compromise and often a difficult intubation.
- Esophageal injury does occur and, as with penetrating neck injury, is not often manifest initially (very high morbidity/mortality if missed).

resuscitation

- Obtain soft-tissue films or CT scan of the neck for clues to the presence of a soft-tissue hematoma and subcutaneous emphysema, and a chest x-ray (CXR) for possible hemopneumothorax.
- Surgical exploration is indicated for:
  - Expanding hematoma
  - Subcutaneous emphysema
  - Tracheal deviation
  - Change in voice quality
  - Air bubbling through the wound
- Pulses should be palpated to identify deficits and thrills, and auscultated for bruits.
- A neurologic exam should be performed to identify brachial plexus and/or central nervous system (CNS) deficits as well as Horner's syndrome.

management

- Most Zone II penetrating injuries require exploration in the OR.
- Injuries to Zones I and III may be taken to OR or managed conservatively using a combination of angiography, bronchoscopy, esophagoscopy, gastrografin or barium studies, and computed tomography (CT) scanning.

WARD TIP

Avoid unnecessary manipulation of the neck as this may dislodge a clot.

WARD TIP

Keep cervical in-line stabilization until C-spine fracture has been ruled out.

WARD TIP

Tracheostomy is the procedure of choice in the patient with laryngotracheal separation.

WARD TIP

Control of hemorrhage in the emergency department is via direct pressure (no blind clamping).

WARD TIP

Never blindly probe a neck wound as this may lead to bleeding in a previously quiescent wound.

EXAM TIP

Beck's tamponade triad:
1. Hypotension
2. JVD
3. Muffled heart sounds

WARD TIP

Clinically apparent tamponade may result from acute accumulation of as little as 60–100 mL of blood in the pericardial space.

OR TIP

1. Pericardium should be opened anterior and parallel to phrenic nerve (longitudinally).
2. Lacerations are repaired with 3-0 nonabsorbable suture and pledgets.
3. A Foley catheter with inflated balloon may be used as a temporizing measure to gain control for large defects.

Spinal Trauma

See Neurosurgery chapter.

Thoracic Trauma

Pericardial Tamponade

- Life-threatening emergency usually seen with penetrating thoracic trauma, but may be seen with blunt thoracic trauma as well.
- Signs include tachycardia, muffed heart sounds, jugular venous distention (JVD), hypotension, and electrical alternans on electrocardiogram (ECG) (see Figure 4-2).
- Diagnosis may be confirmed with cardiac ultrasound (usually as part of focused assessment with sonography for trauma [FAST] exam).
- Requires immediate decompression via needle pericardiocentesis, pericardial window, or thoracotomy with manual decompression.

Blunt Cardiac Trauma

- Usually secondary to motor vehicle collision (MVC), fall from height, crush injury, blast injury, direct violent trauma.
- Screen for blunt cardiac injury with ECG.

Pneumothorax

Definition

Air in the pleural space.

Signs and Symptoms

- Usually asymptomatic.
  - Chest pain
- Dyspnea.
- Hyperresonance of affected side.
- Decreased breath sounds on affected side.

Diagnosis

- Upright CXR is ~83% sensitive and demonstrates an absence of lung markings where the lung has collapsed (see Figure 4-3).
- Lung ultrasound, while operator dependent, is increasingly being used to detect pneumothorax.

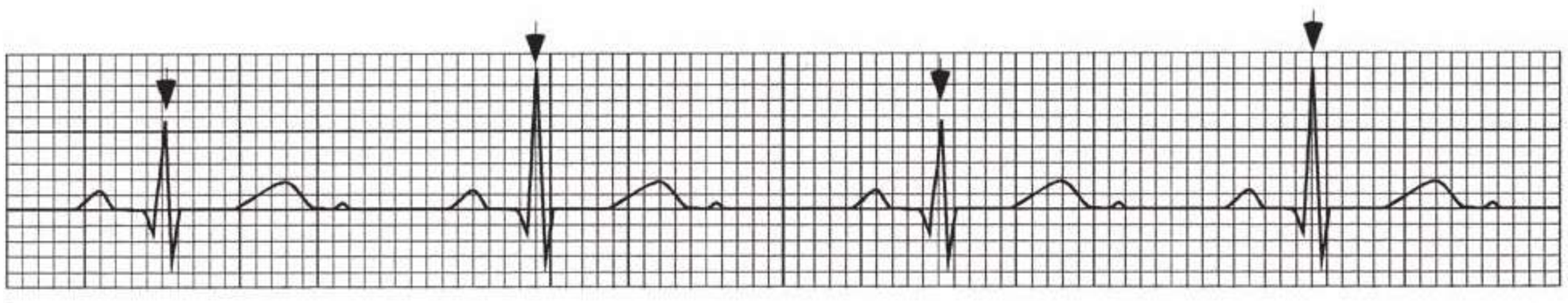
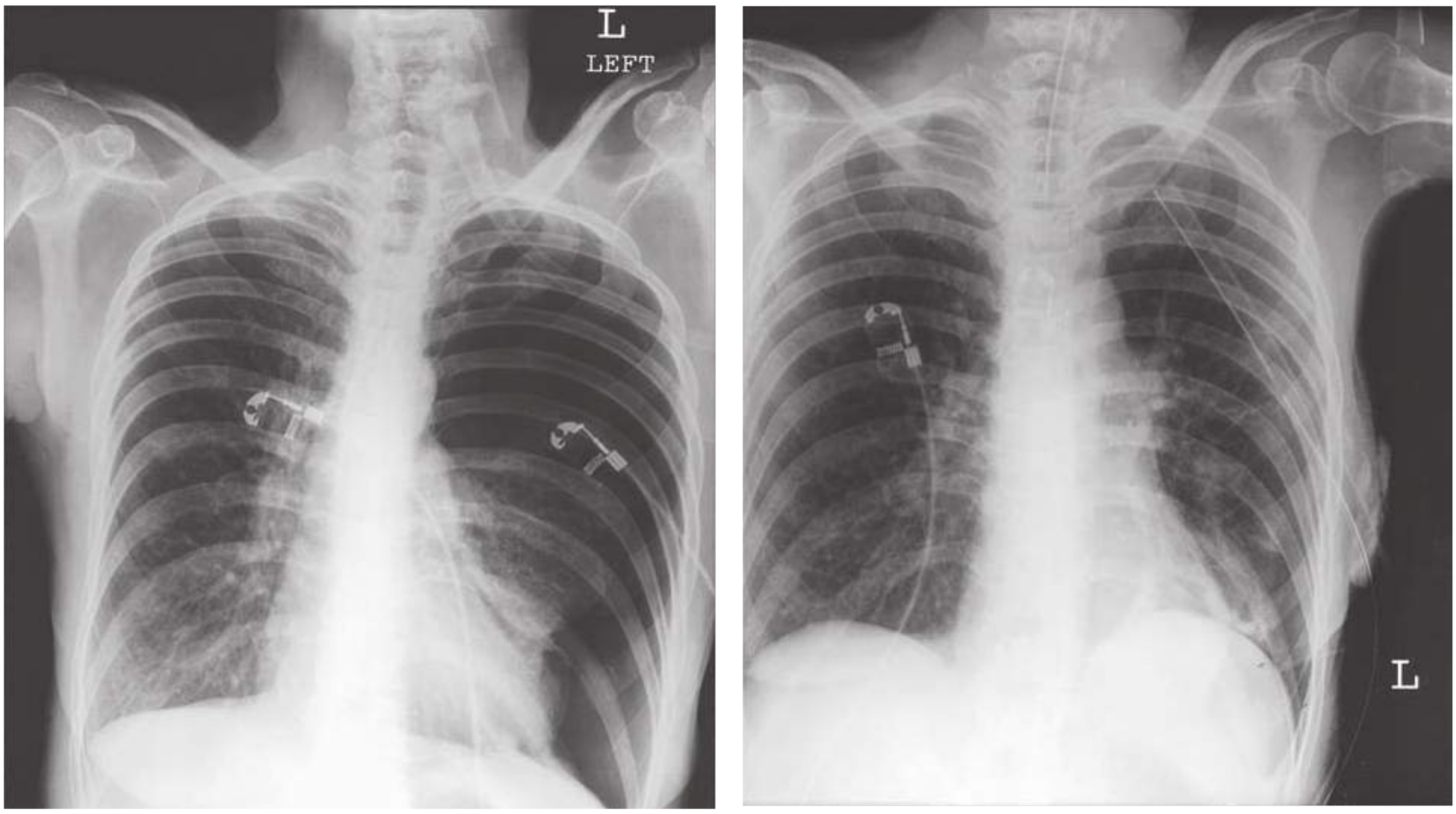


FIGURE 4-2. ECG demonstrating electrical alternans. Note alternating heights of the R in the QRS complexes.



a B

FIGURE 4-3. a. CXr demonstrating left-sided pneumothorax. (Note lack of lung markings.) B. Same patient, after tube thoracostomy and endotracheal intubation.

Treatment

Tube thoracostomy: Chest tube placement should be confirmed by CXR.

Tension Pneumothorax

- Life-threatening emergency caused by air entering the pleural space (most often via a hole in the lung tissue) and unable to escape.
- Causes total ipsilateral lung collapse and mediastinal shift (away from injured lung), impairing venous return and thus decreased cardiac output, eventually resulting in shock.

Signs and Symptoms

Same as for pneumothorax, plus tracheal deviation away from affected side (in tension pneumothorax).

Treatment

- Requires immediate needle decompression followed by tube thoracostomy.
- Needle decompression involves placing a needle or catheter over a needle into the second intercostal space, midclavicular line, over the rib on the side of the tension pneumothorax, followed by tube thoracostomy (chest tube).

Hemothorax

The presence of blood in the chest.

- More than 200 cc of blood must be present before blunting of costophrenic angle will be seen on CXR.
- Treatment involves chest tube placement and drainage, and control of bleeding.

EXAM TIP

Typical scenario: A 19-year-old male who was stabbed in the chest with a knife presents complaining of dyspnea. Breath sounds on one side are absent. Think: Pneumothorax.

WARD TIP

The neurovascular bundle runs on the inferior margin of each rib.

WARD TIP

When the clamp enters the pleura, a rush of air or fluid should be obtained.

WARD TIP

Size of chest tube to use:

- For adult large hemothorax: 36–40 French.
- For adult pneumothorax: 24 French or pigtail catheter.
- For children: Four times the size of appropriate endotracheal tube size:

$$\text{ET tube size} = \frac{\text{age} + 16}{4}$$

ExAm TIP

A diagnosis of tension pneumothorax via x-ray is a missed diagnosis. Do not delay treatment of a suspected tension pneumothorax in order to confirm your suspicion (i.e., tension pneumothorax is a clinical diagnosis).

WARD TIP

Remember to confirm appropriate chest tube placement with CXR.

WARD TIP

The rule of 1/3's for abdominal stab wounds

- 1/3 will not penetrate the fascia
- 1/3 will penetrate the fascia but not cause injuries
- 1/3 will penetrate the fascia and cause intraabdominal injuries

WARD TIP

- One fourth of hemothorax cases have an associated pneumothorax.
- Three fourths of hemothorax cases are associated with extrathoracic injuries.

Indications for Thoracotomy

- 1,500 cc initial drainage from the chest tube.
- 200 cc/hr for 4 hours continued drainage:
  - Thoracic great vessel injury.
  - Esophageal injury.
  - Patients who decompensate after initial stabilization.

Diagnostic Modalities

- Angiography to localize injury and plan appropriate operation.
- CT scan for patients with normal initial CXR but suspicious mechanism and requiring CT for other reasons. If CT identifies injury, angiography still required for precise delineation of injury.
- Transesophageal echocardiogram (TEE):
  - Fast, no contrast required, concurrent evaluation of cardiac function, versatile in terms of location.
  - Contraindicated if potential airway problem or C-spine injury.
  - Not as sensitive or specific as angiography or CT scan.
  - User dependent.

Definitive Treatment

- Surgical: Control bleeding, reconstruction, with graft if needed.
- Control hypertension pharmacologically.
- Endovascular stenting.
- Cardiac bypass as needed.
- Nonoperative: Close observation and pharmacologic treatment.

Abdominal Trauma

General

Penetrating abdominal injuries (PAIs; see Figure 4-4) resulting from a gunshot wound create damage via three mechanisms:

1. Direct injury by the bullet itself.
2. Injury from fragmentation of the bullet.
3. Indirect injury from the resultant "shock wave."

PAIs resulting from a stabbing mechanism are limited to the direct damage of the object of impalement.

Blunt abdominal injuries (BAIs) also have three general mechanisms of injury:

1. Injury caused by the direct blow.
2. Crush injury.
3. Deceleration injury.

Physical examination

Signs

- **Seat-belt sign**—ecchymotic area found in the distribution of the lower anterior abdominal wall and can be associated with perforation of the bladder or bowel as well as a lumbar distraction fracture (Chance fracture).

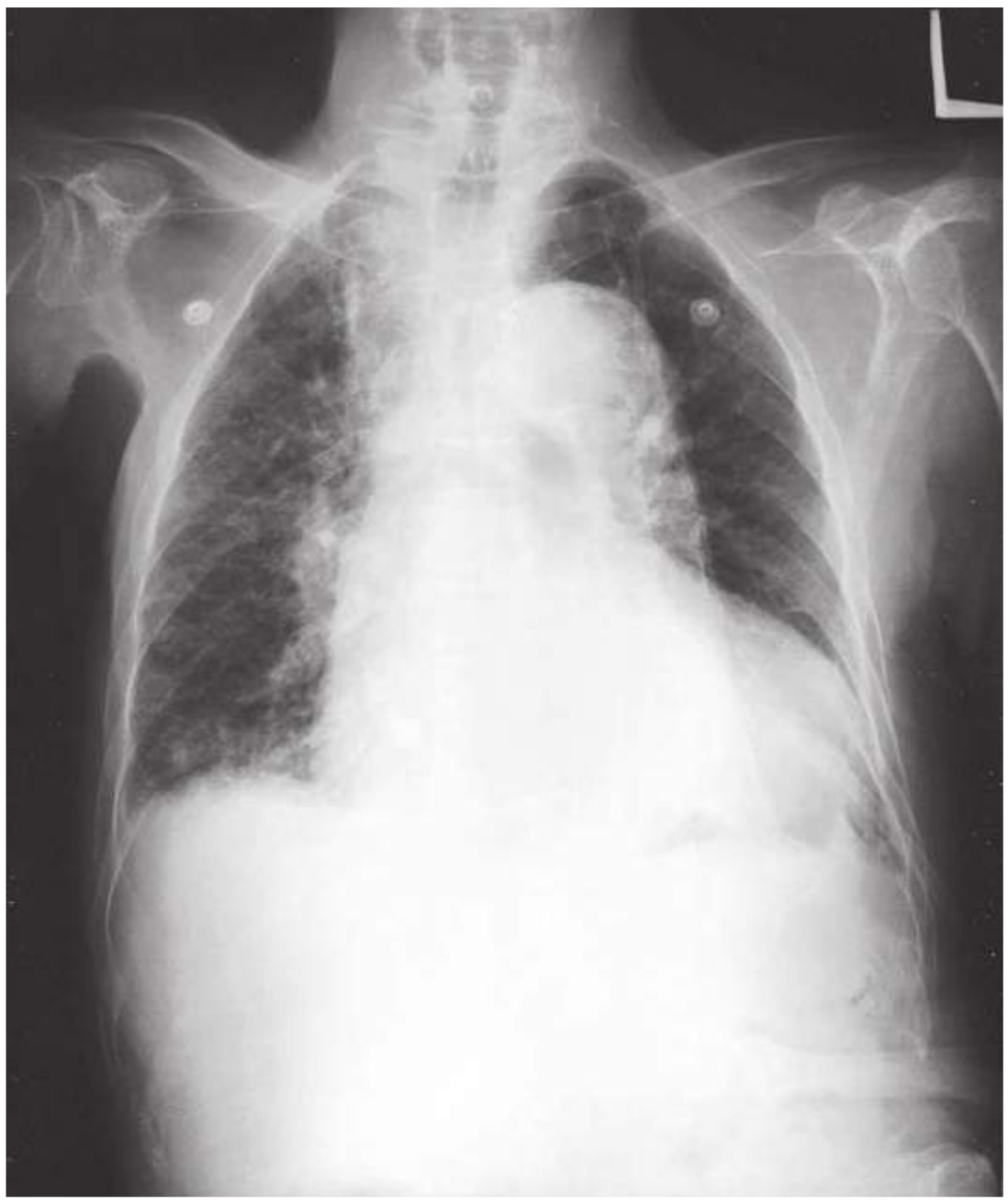


FIGURE 4-4. CXr illustrating wide mediastinum due to penetrating trauma of the ascending aorta.

- **Cullen's sign** (periumbilical ecchymosis) is indicative of intraperitoneal hemorrhage.
- **Grey-Turner's sign** (flank ecchymoses) is indicative of retroperitoneal hemorrhage.
- **Kehr's sign**—left shoulder or neck pain secondary to splenic rupture. It increases when patient is in Trendelenburg position or with left upper quadrant (LUQ) palpation (caused by diaphragmatic irritation).

General

- Inspect the abdomen for evisceration, entry/exit wounds, impaled objects, and a gravid uterus.
- Check for tenderness, guarding, and rebound.

Diagnosis

- Perforation: AXR and CXR to look for free air.
- Diaphragmatic injury: CXR to look for blurring of the diaphragm, hemothorax, or bowel gas patterns above the diaphragm (at times with a gastric tube seen in the left chest).

FOCUSED ABDOMINAL SONOGRAPHY FOR TRAUMA (FAST)

- Positive if free fluid is demonstrated in the abdomen.
- Has largely replaced DPL in most trauma centers.

WARD TIP

Bleeding usually stops spontaneously for low-velocity gunshot wounds and most stab wounds.

WARD TIP

Potential causes of iatrogenic great vessel injury:

- Central venous pressure (CVP) line or chest tube placement
- Intra-aortic balloon pump (IABP) placement
- Over inflation of Swan–Ganz balloon

WARD TIP

Traumatic aortic rupture is a high-mortality injury: Almost 90% die at the scene, and another 50% die within 24 hours.

EXAM TIP

Injury to innominate or subclavian arteries will result in absent or decreased upper extremity pulses and blood pressure (BP) with increased lower extremity BP.

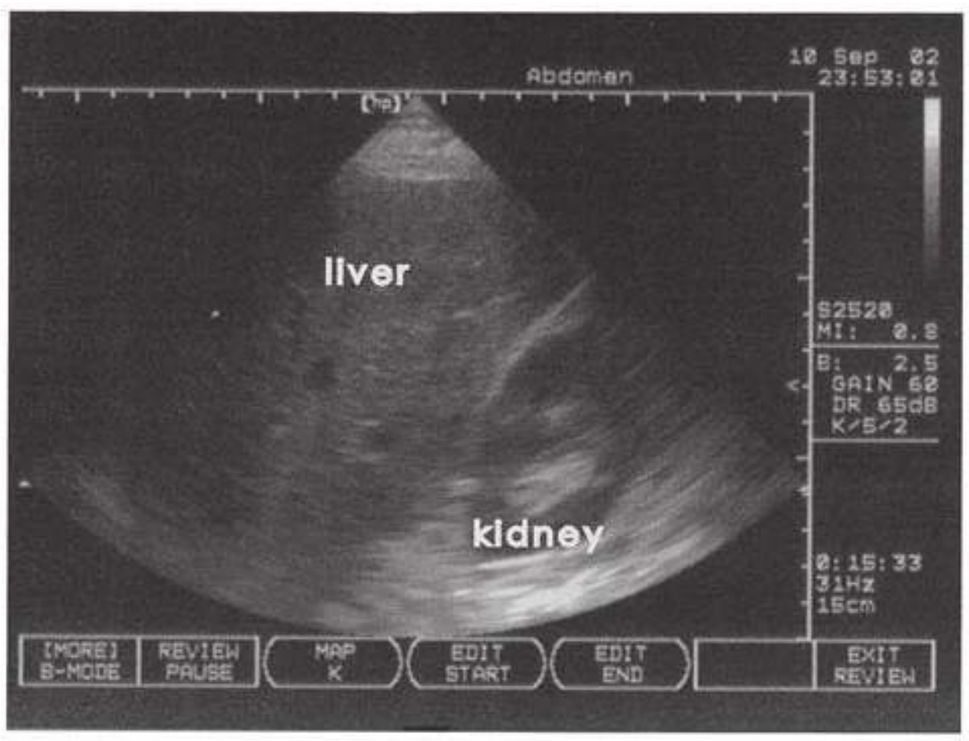
- Four views are utilized to search for free intraperitoneal fluid (presumed to be blood in the trauma victim) that collects in dependent areas and appears as hypoechoic areas on ultrasound (see Figures 4-5 and 4-6):
  - Morrison's pouch (RUQ): Free fluid can be visualized between the liver and kidney.
  - Splenorenal recess (LUQ): Free fluid can be visualized between the spleen and kidney.
  - Pouch of Douglas lies above the rectum (probe is placed in the supra-pubic region).
  - Subxiphoid and parasternal views to look for hemopericardium.

Advantages

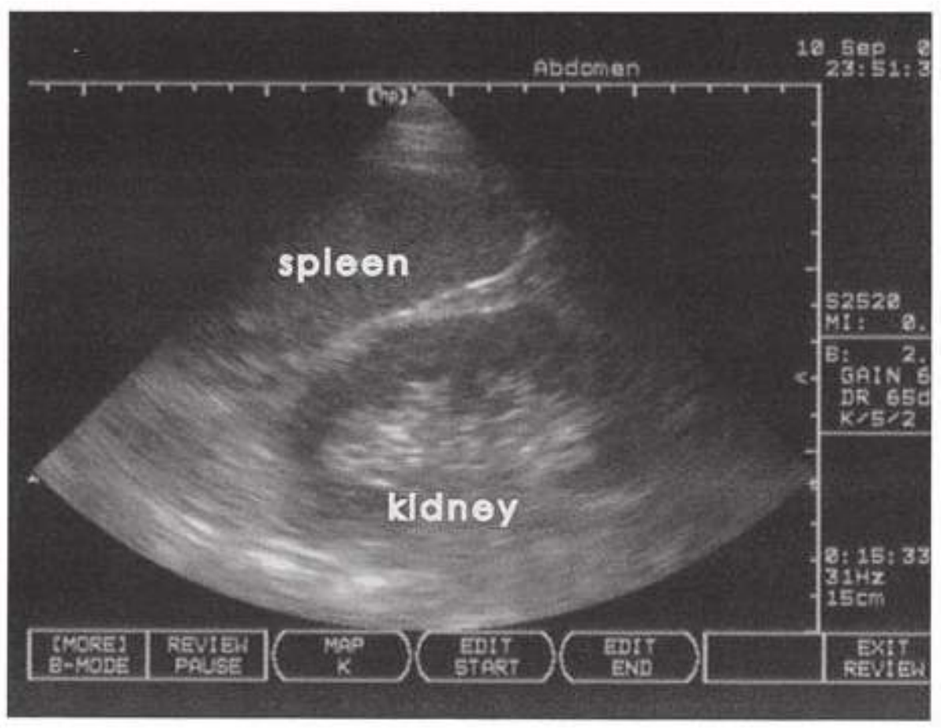
- A rapid bedside screening study.
- Noninvasive.
- Not time consuming, can be repeated if necessary.
- Eighty to 95% sensitivity for intra-abdominal blood.

Disadvantages

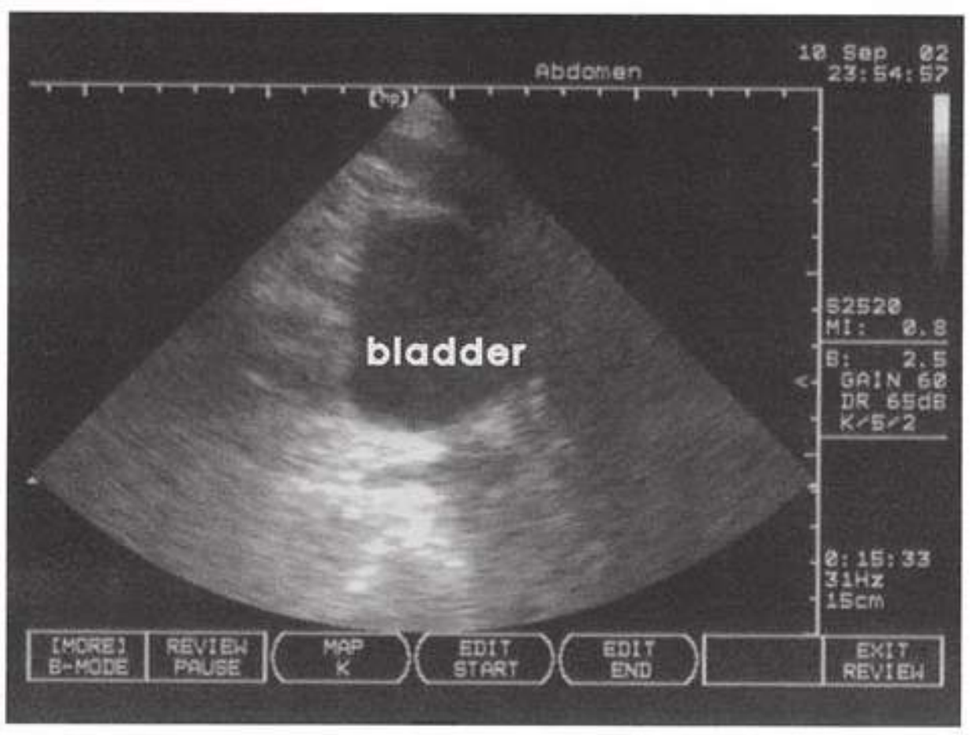
- Operator dependent.
- Low specificity for individual organ injury.



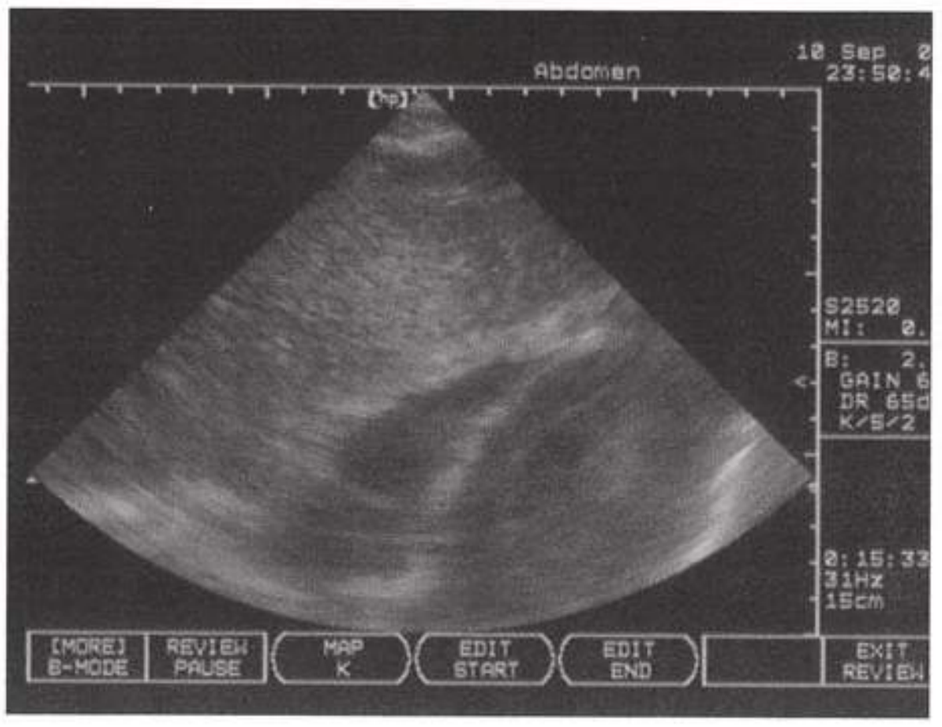
a



B

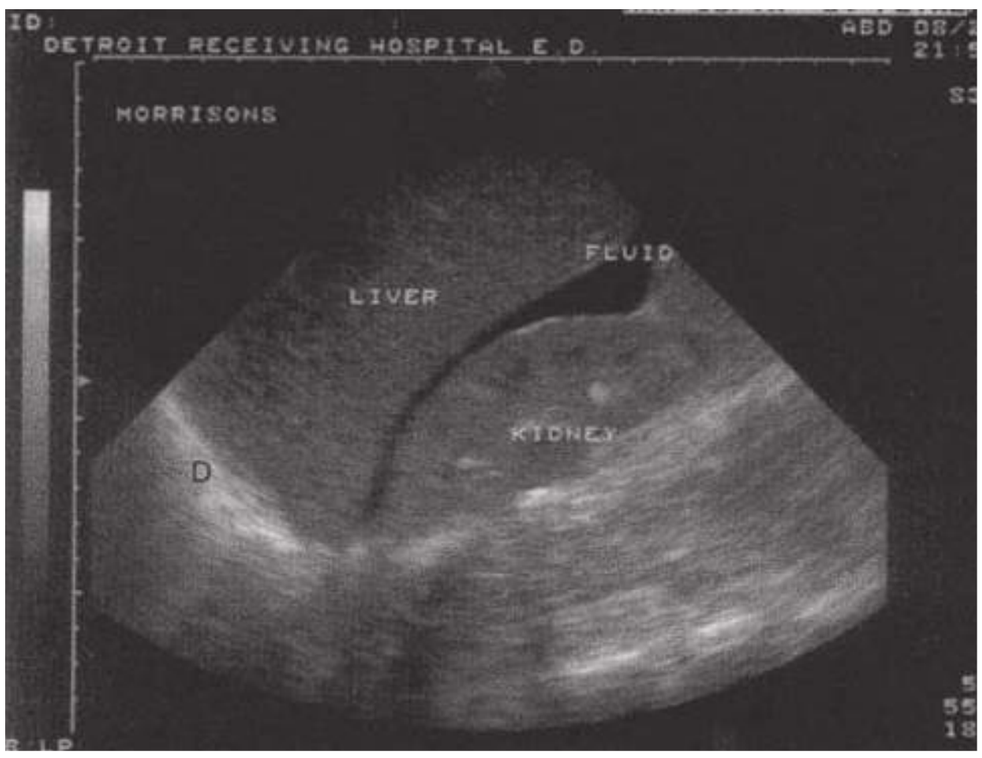


C

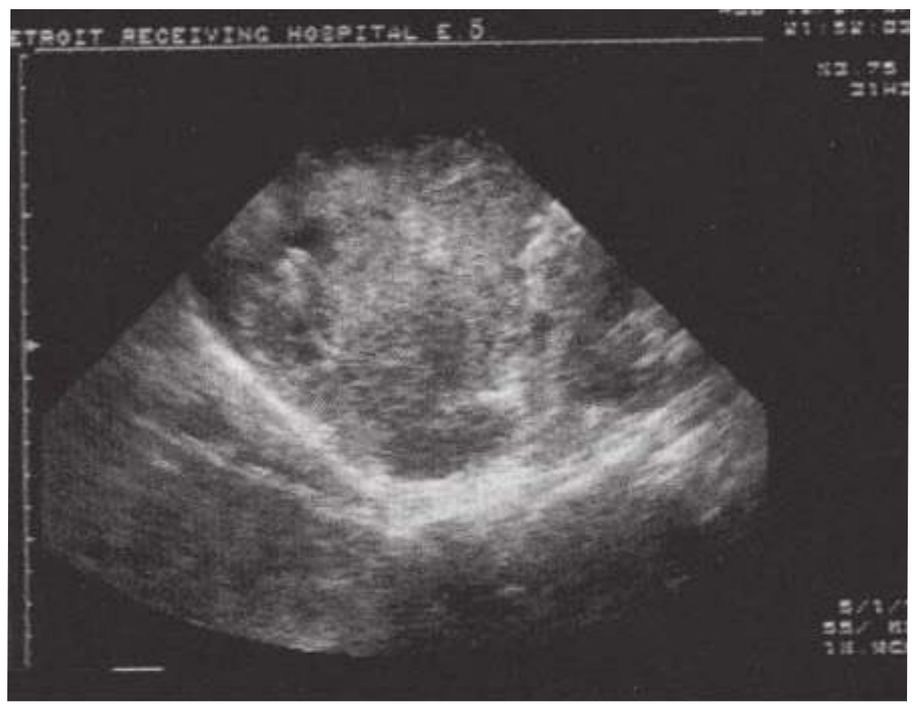


D

FIGURE 4-5. Normal focused sonography for trauma (FAST) views. A. Liver–kidney view. **B.** Spleen–kidney interface. **C.** Bladder view. **D.** Pericardial view.



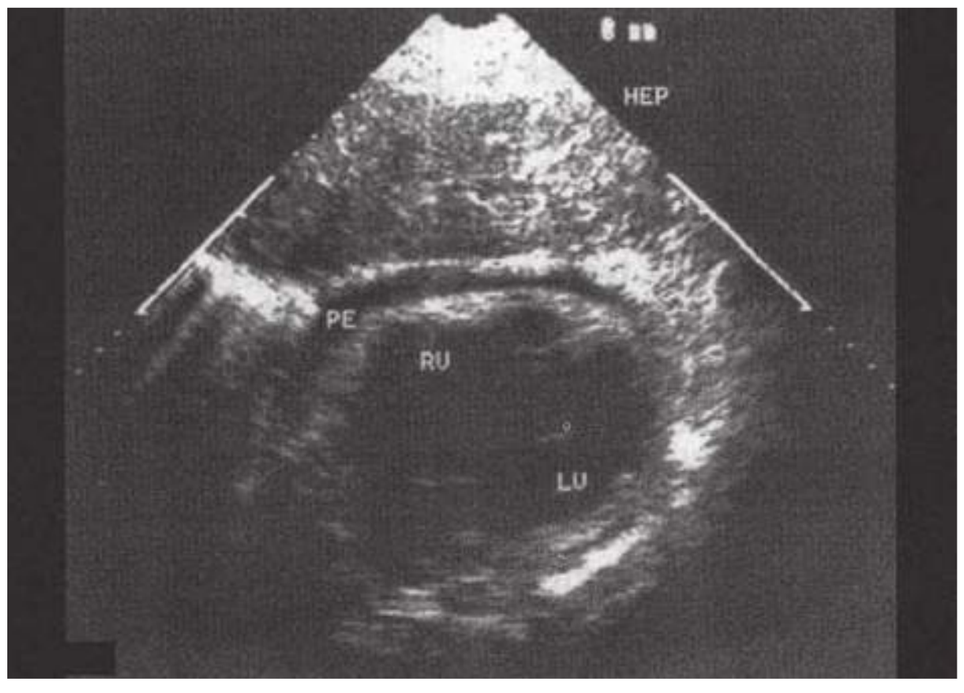
a



B



C



D

FIGURE 4-6. abnormal FaST views. A. Liver–kidney view demonstrating presence of fluid (black stripe) in Morrison's pouch compared to Figure 4-5A. **B.** Fluid in splenoral recess. **C.** Free fluid in the pelvis. **D.** Pericardial effusion (denoted as PE). (A, B, and D reproduced, with permission, from Jones RA, Welch R, Falcone R. Handbook of Trauma Ultrasonography. 1st ed. Bloomfield Hills, MI: Haines International; September 1999. C reproduced, with permission, from Melanson SW, Heller M. The emerging role of bedside ultrasonography in trauma care. Emerg Med Clin North Am 1998 Feb; 16(1):165-89.)

diagnostic peritoneal lavage (dpl)

Open DPL

Similar to open port placement in laparoscopic surgery (peritoneal cavity is entered under direct vision) using the Hassan port.

Closed DPL

Using the Seldinger technique, a catheter is placed through the needle and advanced into the peritoneum. The needle placement is similar to the closed technique of port placement in laparoscopy using the Veress needle.

advantages

- Performed at bedside.
- Widely available.
- Highly sensitive for hemoperitoneum.
- Rapidly performed.

OR TIP

- Patient should be prepped and draped from sternum to knees to allow alternate access from the groin in the event of an emergency.
- Traditional approach (i.e., left anterolateral sternotomy) is used for the unstable patient with an undiagnosed injury.
- Angiography in the stable patient may dictate an alternate operative approach based on the location of injury.

Disadvantages

- Invasive.
- Risk for iatrogenic injury (< 1%).
- Low specificity (many false positives).
- Does not evaluate the retroperitoneum.

CT Scanning

- Useful for the hemodynamically stable patient.
- Has a greater specificity than DPL and ultrasound (US).
- Noninvasive.
- Relatively time consuming when compared with FAST.

Angiography

- May be used to identify and embolize pelvic arterial bleeding secondary to pelvic fractures, or to assess blunt renal artery injuries diagnosed by CT scan.
- Otherwise limited use for abdominal trauma.

Serial Hematocrits

Serial hematocrits (every 4–6 hours) should be obtained during the observation period of the hemodynamically stable patient.

Laparoscopy

- Usage is increasing (mainly to identify peritoneal penetration from gunshot/knife wound), especially for the stable or marginally stable patient who would otherwise require a laparotomy.
- Helpful for evaluation of diaphragm.
- May help to decrease negative laparotomy rate.
- However, may miss hollow organ injuries.

Indications for Exploratory Laparotomy

- Abdominal trauma and hemodynamic instability.
- Evisceration.
- Peritonitis.
- Diaphragmatic injury (see Figure 4-7).
- Hollow viscus perforation: Free intraperitoneal air.
- Intraperitoneal bladder rupture (diagnosed by cystography).
- Positive DPL.
- Surgically correctable injury diagnosed on CT scan.
- Removal of impaled weapon.
- Rectal perforation.
- Transabdominal missile (bullet) path (e.g., a gunshot wound to the buttock with the bullet being found in the abdomen or thorax).

Types of injury: General Approach

liver injury

See Hepatobiliary System chapter.

OR TIP

Safe margin for crossclamping of aorta to control bleeding is < 30 minutes.

Exam TIP

Typical scenario: A 25-year-old female presents after a high-speed MVC with dyspnea and tachycardia. There is local bruising over right side of her chest. CXR shows a right upper lobe consolidation. Think: Pulmonary contusion.

WARD TIP

The most frequently injured solid organ in penetrating trauma is the liver.

WARD TIP

The most frequently injured solid organs in blunt trauma are the liver and spleen.

Exam TIP

Peritonitis and guarding in a neurologically intact patient obviate the need for much diagnostic workup. Trauma laparotomy is indicated in this setting.

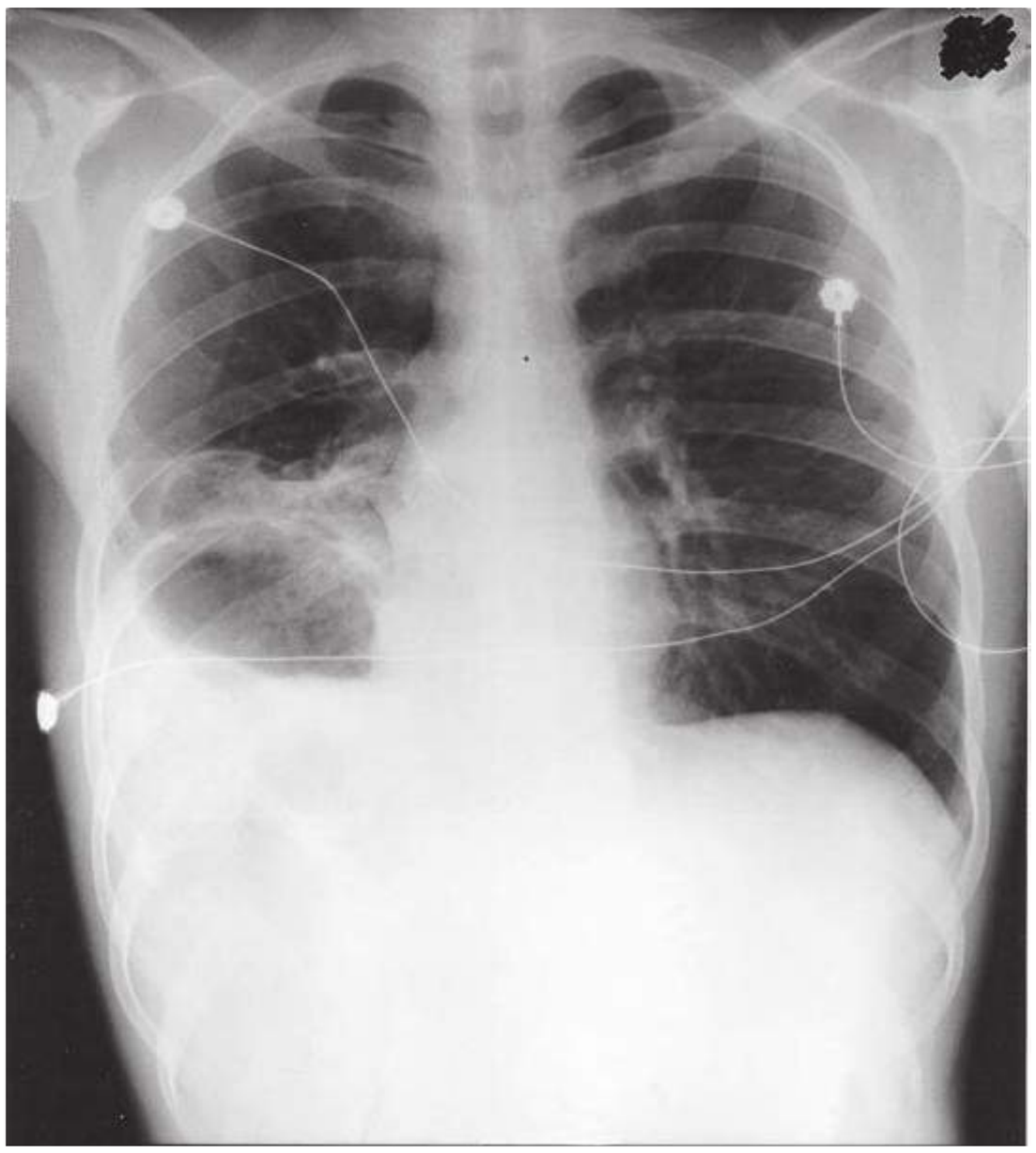


FIGURE 4-7. CXr demonstrating ruptured diaphragm. Note elevation and irregular contour of diaphragm, and viscera in chest.

SPleniC Trauma

See Spleen chapter.

Bowel injury

Stomach, Jejunum, and Ileum

- Isolated leaks from penetrating trauma lead to minimal contamination and patients usually do well if diagnosis is not delayed.
- Blunt injuries are "blowouts" resulting frequently from lap belts, and occur near the ligament of Treitz and the ileocecal valve.
- Mesentery can be significantly injured following blunt trauma.
- **Diagnosis**:
  - If the patient is awake and reliable, the exam is important to look for peritoneal irritation.
  - If the exam is not reliable, DPL or laparoscopy may be required.
  - CT scan has a high false-negative rate for small bowel injuries.
  - Look for free air on upright CXR.
  - Laparotomy for gastric or small bowel injury with primary repair and peritoneal lavage except in cases that have heavy soiling of the peritoneal cavity and present late, where intestinal diversion must be considered (e.g., ileostomy).
    - Small bowel resection is needed where more than 50% of the bowel circumference is transected or several penetrating injuries are present within a very short segment of bowel where resecting is a better option than individually repairing each hole.

EXAM TIP

In a stable patient with neurologic dysfunction, whether from drugs, alcohol, head trauma, or baseline dementia, exam findings have a limited ability to direct care. These patients often require additional diagnostic tests.

WARD TIP

Serial abdominal examinations should be performed.

WARD TIP

DPL should be undertaken only after gastric and urinary decompression.

WARD TIP

Criteria for a positive DPL:

- > 10 mL gross blood on initial aspiration.
- Presence of bile, stool, or intestinal contents.
- > 100,000 red blood cells (RBCs).
- > 500 white blood cells (WBCs).
- Gram stain with bacteria or vegetable matter.
- Amylase > 20 IU/L.

WARD TIP

Absolute contraindication for DPL: Clear indication for laparotomy (e.g., peritonitis).

ExAm TIP

CT is the most sensitive test for retroperitoneal injury.

WARD TIP

Contrast use:

- PO contrast to assess location and integrity of upper gastrointestinal (GI) tract.
- IV contrast to look for organ or vascular injury.
- Contrast enema to evaluate the colon and rectum.

Duodenum

- **Mechanisms:** Three fourths of injuries result from penetrating trauma.
- **Diagnosis:**
  - Upper GI series with water-soluble contrast.
  - CT and DPL often miss duodenal injuries.
- **Treatment:**
  - Eighty percent of patients are able to undergo a primary repair.
  - Repair may be protected with an omental patch, jejunal serosal patch, and/or gastric diversion.
  - More complex injuries need pyloric exclusion or rarely pancreaticoduodenectomy (Whipple procedure).

Large Bowel

- Injuries generally occur via a penetrating mechanism (75% gunshot wound, 25% stab wound). Blunt injuries are rare but result from MVCs. Iatrogenic transanal injuries may also occur.
- **Signs and symptoms:** Abdominal distention, tenderness, guaiac-positive stool.
- **Diagnosis:**
  - In an awake and reliable patient, exam findings are consistent with peritonitis.
  - CXR may show free air.
  - In a patient with a flank injury but without clear peritoneal signs, consider a contrast enema.
- **Treatment:**
  - Primary repair: For small or medium-sized perforations, repair the perforation or, if needed, resect the affected segment and close with primary anastomosis. A proximal diverting stoma (e.g., ileostomy) is commonly placed.
  - Anastomosis is contraindicated in the setting of massive soiling.

rectum

- Two thirds are extraperitoneal.
- **Mechanism:** Majority by gunshot injury.
- **Diagnosis:**
  - DRE/guaiac: Suspicion increased by blood in stool or palpation of defect or foreign body on exam.
  - Rigid proctoscopy: May be done in OR if needed; mandatory for patients with known trajectory of knife or gunshot wound across pelvis or transanal; if patient is unstable, may be delayed until after resuscitation.
  - X-ray to look for missiles or foreign bodies.
- **Treatment:**
  - Diversion via colostomy is key.
  - Extraperitoneal injuries must be diverted via colostomy but may not need to be repaired (if not too big and not easily accessible).
  - Colostomy may be closed in 3–4 months.

anus

- Reconstruct sphincter as soon as patient is stabilized.
- Divert with sigmoid colostomy.

Pancreatic Injury

General

- **Mechanism:** Largely penetrating (gunshot wound > stab wound).
- Seventy-five percent of patients with penetrating injury to the pancreas will have associated injuries to the aorta, portal vein, or inferior vena cava.

Diagnosis

- Inspect pancreas during laparotomies performed for other indications.
- Check amylase and lipase.
- CT: Look for parenchymal fracture, intraparenchymal hematoma, lesser sac fluid, fluid between splenic vein and pancreatic body, retroperitoneal hematoma or fluid.
- Endoscopic retrograde cholangiopancreatography (ERCP): May be used in the stable patient if readily available or available intraoperatively; also may be used to evaluate missed injuries.

Treatment

- Nonoperative: May follow with serial labs and exam if patient can be reliably examined.
- Operative:
  - No ductal injury: Hemostasis and external drainage.
  - Distal transection, parenchymal injury with ductal injury: Distal pancreatectomy with duct ligation.
  - When duodenum or pancreatic head is devitalized, consider Whipple or total pancreatectomy.
  - Proximal transection/injury with probable ductal disruption:
    - If duct is spared, external drainage.
    - If duct is damaged, external drainage and pancreatic duct stenting may be considered.

OR TIP

Trauma laparotomy:
- Midline incision.
- Pack all four quadrants with laparotomy pads.
- Evacuation of gross blood and clot.
- Control bleeding.
- Resuscitate as needed.
- Systematically remove pads and inspect for source(s) of injury.
- Definitive repair based on stability of patient and type of injury.

EXAM TIP

Any wound from the nipple line to the gluteal crease can cause peritoneal or retroperitoneal injury.

OR TIP

Have a low threshold for conversion to laparotomy during laparoscopy.

Genitourinary (GU) Trauma

General

- Often overlooked in the initial evaluation of the multiply injured trauma patient.
- Diagnostic evaluation of the GU tract is performed in a "retrograde" fashion (i.e., work your way back from the urethra to the kidneys and renal vasculature).

Signs and Symptoms

- Flank or groin pain.
- Blood at the urethral meatus.
- Ecchymoses on perineum and/or genitalia.
- Evidence of pelvic fracture.
- Rectal bleeding.
- A "high-riding" or superiorly displaced prostate.

EXAM TIP

Because of a common mechanism, Chance fractures and blunt small bowel injury are strongly associated. If one is present you should look for the other.

OR TIP

To determine viability of the bowel in the OR, inject fluorescein dye IV, and use a Wood's lamp to inspect the bowel. Nonviable bowel will have patchy or no fluorescence.

WARD TIP

Duodenal hematoma may result from an MVC but has been found to be associated with child abuse in the pediatric population. Patients present with signs and symptoms of small bowel obstruction and require CT/upper GI series for diagnosis. Treatment is nonoperative and includes NGT decompression total parental nutrition (TPN) and reevaluation with upper GI series after about 1 week.

EXAM TIP

Thirty percent of patients with rectal injury will have an associated injury to the bladder.

EXAM TIP

Minimizing the time from injury to treatment is important in minimizing morbidity and mortality associated with pancreatic injury.

WARD TIP

Eighty percent of pancreas can be resected without endocrine or exocrine dysfunction.

WARD TIP

Suspect GU trauma with:

- Straddle injury: Pelvic fracture.
- Penetrating injury to lower abdomen.
- Falls from height.
- Hematuria noted on Foley insertion.

Urinalysis

- The presence of gross hematuria indicates GU injury and often concomitant pelvic fracture.
- Urinalysis should be done to document presence or absence of microscopic hematuria.
- Microscopic hematuria is usually self-limited.

retrograde Urethrogram

- Should be performed in any patient with suspected urethral disruption (before Foley placement).
- A preinjection KUB (kidneys, ureters, bladder) film should always be taken.

retrograde Cystogram

- Should be performed on patients with gross hematuria or a pelvic fracture.
- Extravasation of contrast into the pouch of Douglas, paracolic gutters, and between loops of intestine is diagnostic for intraperitoneal rupture.
- Extravasation of contrast into the paravesicular tissue or behind the bladder is indicative of extraperitoneal bladder rupture.

Bladder rUpture

Intraperitoneal

- Usually occurs secondary to blunt trauma to a full bladder.
- Treatment is surgical repair.

Extraperitoneal

- Usually occurs secondary to pelvic fracture.
- Treatment is nonsurgical management by Foley drainage.

Ureteral injUry

- Least common GU injury (mostly iatrogenic).
- Must be surgically repaired.
- Diagnosed at the time of IVP or CT scan during the search for renal injury.

renal injUry

- Commonly diagnosed by CT scan with contrast.
- Grade IV and V operative; the rest conservative (see Table 4-1 and Figure 4-8).

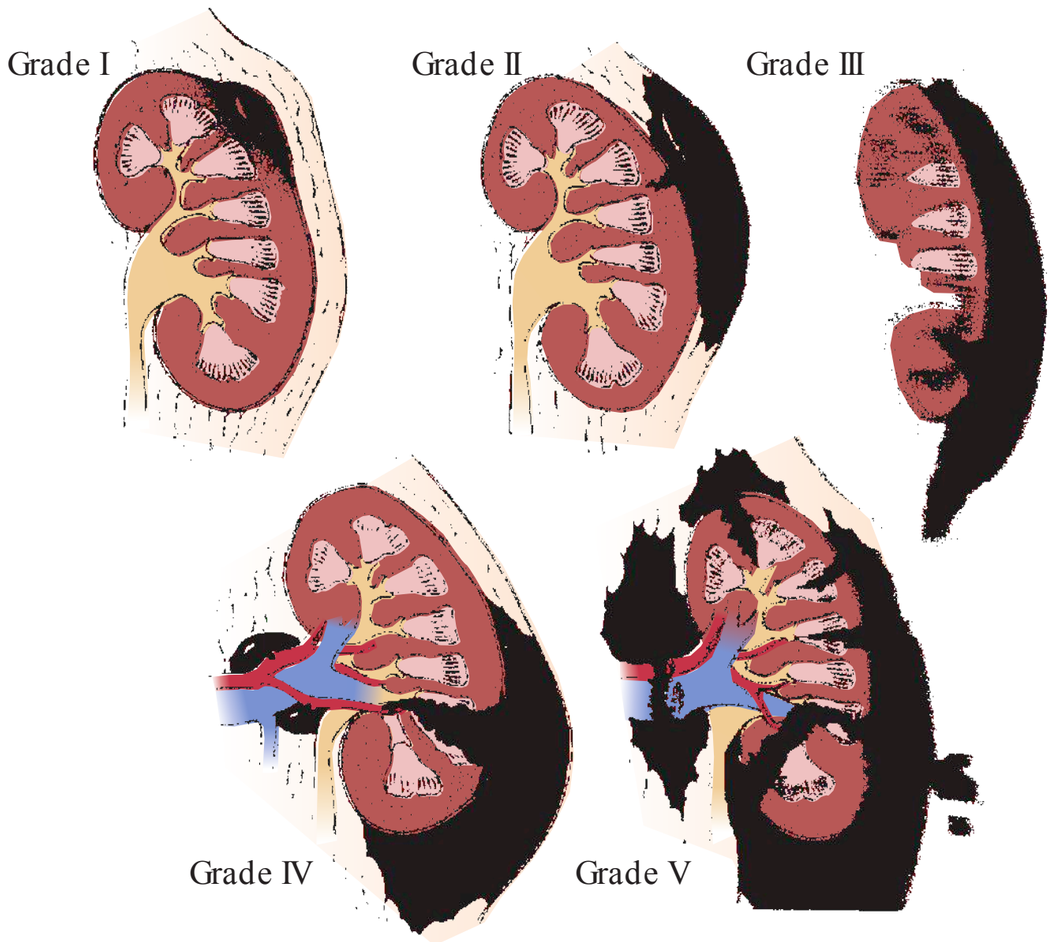


FIGURE 4-8. Organ injury scaling system for renal trauma. (Reproduced, with permission, from Feliciano DV, Mattox KL, Moore EE. Trauma. 6th ed. New York: McGraw-Hill; 2008: Fig. 39-6).

TABLE 4-1. urologic Injury Scale of the american association for the Surgery of Trauma

Grade		Injury description
renal Injury Scale		
I	Contusion	Microscopic or gross hematuria; urologic studies normal
	Hematoma	Subcapsular, nonexpanding without parenchymal laceration
II	Hematoma	Nonexpanding perirenal hematoma confned to the renal retroperitoneum
	Laceration	< 1 cm parenchymal depth of renal cortex without urinary extravasation
III	Laceration	> 1 cm parenchymal depth of renal cortex without collecting-system rupture or urinary extravasation
IV	Laceration	Parenchymal laceration extending through the renal cortex, medulla, and collecting system
	Vascular	Main renal artery or vein injury with contained hemorrhage
V	Laceration	Completely shattered kidney
	Vascular	Avulsion of renal hilum that devascularizes kidney

Modifed, with permission, from Feliciano DV, Mattox KL, Moore EE. Trauma. 6th ed. New York: McGraw-Hill; 2008: Table 39-1.

WARD TIP

Blood at the urethral meatus is virtually diagnostic for urethral injury and demands early retrograde urethrogram before Foley placement.

WARD TIP

Do not probe perineal lacerations as they are often a sign of an underlying pelvic fracture and disruption of a hematoma may occur.

EXAM TIP

Signs of arterial insufficiency: The 6 Ps
Pain
Pallor
Paresthesias
Pulse deficit
Poikilothermia
Paralysis

WARD TIP

Rhabdomyolysis causes myoglobin release, which can cause renal failure. Maintaining a high urine output together with alkalinization of the urine can help prevent the renal failure by reducing precipitation of myoglobin in the kidney.

Critical Care

Shock 62
- Definition 62
- Pathophysiology and Types of Shock 62
- Hypovolemic Shock—Reduced Venous Return, Adequate Pump Function 62
- Distributive Shock 63
- Anaphylactic Shock 63
- Neurogenic Shock 65
- Cardiogenic Shock 65
- Obstructive Shock 66

Swan–Ganz Catheter 66

Pressors and Inotropes 68
- Effects and Side Effects 68
- Dobutamine 68
- Isoproterenol 68
- Milrinone 69
- Dopamine 69
- Norepinephrine 69
- Epinephrine 69
- Phenylephrine 70

Mechanical Ventilation 70
- Setting the Ventilator 70
- Adjusting the Ventilator 70

Acute Respiratory Distress Syndrome (ARDS) 71
- Causes 72
- Management 72

Shock

Definition

Shock is defined as inadequate tissue perfusion. The delivery of oxygen and nutrients is not sufficient to maintain normal tissue and cellular function.

Shock becomes irreversible if not treated early.

WARD TIP

Think ABCs with any patient in "shock": Secure airway, breathing, and circulation.

PathoPhysiology anD tyPes of shock

Shock describes a state of imbalance between tissue substrate delivery (supply) and tissue substrate consumption (demand).

Tissue perfusion is determined by:

1. Cardiac Output (CO) = Stroke Volume (SV) × Heart Rate (HR)
2. Systemic Vascular Resistance (SVR) = [Mean Arterial Pressure (MAP) - Central Venous Pressure (CVP)]/ [CO]
3. BP = CO × SVR

Deterioration in any one of these factors can cause hypotension and shock. Shock is classified into different types according to which of these factors are abnormal. The four major types of shock are **hypovolemic, distributive (includes septic, anaphylactic, and neurogenic), cardiogenic**, and **obstructive**.

Note: The first step in treatment of any type of shock is control of airway and breathing, next is restoration of circulation (ABC).

WARD TIP

If the skin is warm, it is distributive shock. If the skin is cold and clammy, it is hypovolemic, obstructive, or cardiogenic shock. Shock with bradycardia is neurogenic unless proved otherwise.

WARD TIP

Shock in trauma or postop patient is assumed to be hypovolemic until proven otherwise.

hyPovolemic shock—ReDuceD venous RetuRn, aDequate PumP function

Definition

Decreased tissue perfusion secondary to rapid volume/blood loss (i.e., preload). **CO is consequently decreased**. The causes include bleeding, vomiting/diarrhea, and third spacing (e.g., from burns, bowel obstruction, pancreatitis, surgery).

EXAM TIP

- First sign of hypovolemic shock: tachycardia!

Signs and Symptoms

Early on, patients will have tachycardia, orthostatic hypotension, and cool skin. As the condition progresses, they are hypotensive, have decreased pulse pressure (diastolic blood pressure becomes elevated), become confused, and have cold, clammy skin due to "clamping down" of peripheral vessels via increased sympathetic tone.

WARD TIP

Of the vital organs, the first "casualty" of hypovolemic or cardiogenic shock (both "cold shocks") is the kidneys, as blood is shunted away from the constricted renal arteries. Therefore, it is crucial to monitor for renal failure. An adequate urine output is one of the crucial signs that the treatment is adequate.

Classification of Severity of Hypovolemic Shock

Class I—Compensated: Loss of < 15% of circulating blood volume. Little or no clinical manifestations.

Class II—Partially compensated: Loss of 15–30% of blood volume. Manifestations include mild tachycardia, tachypnea, anxiety, orthostatic hypotension, decreased pulse pressure, and oliguria. Reduced splanchnic and renal blood flow.

Class III—Uncompensated: Loss of 30–40% of blood volume. Hypotension, oliguria, marked tachycardia, and confusion.

Class IV—Life threatening: Loss of > 40% of circulating blood volume. All of the above plus lethargy, mental status change, severe hypotension, and oliguria or anuria.

Treatment

- Rapid initial FLUID resuscitation! Crystalloids (normal saline [NS]/lactated Ringer's [LR]) infusion via two large-bore peripheral IVs is best for volume repletion.
  - Normal saline vs. lactated Ringer's: Large-volume NS infusion may result in hyperchloremic acidosis. Therefore, LR (containing alternative anions to Cl^-) is the preferred choice.
  - Colloid vs. isotonic crystalloid: No evidence that colloid fluids improve mortality in critically ill patients.
- Class III and IV shock: Think about blood transfusion.
- Replacement of blood if hemorrhage is cause.
- Treat underlying cause (i.e., surgical correction if patient has ongoing hemorrhage). Failure to respond to fluid resuscitation is usually due to persistent massive hemorrhage, hence requiring emergent surgical procedure.

WARD TIP

Tachycardia is the first symptom in hypovolemic shock.
Factors that suppress the tachycardic response to hypovolemia:
- Beta blockers
- Athletes
- Damage to autonomic nervous system (as in spinal shock)
- Never use dextrose-containing solution for resuscitation.

Distributive shock

Definition

A family of shock states that are caused by systemic vasodilation (i.e., severe decrease in SVR). They include septic shock (most common), neurogenic shock, and anaphylactic shock. These patients will have warm skin from vasodilation.

Septic Shock

Infection that causes vessels to dilate and become leaky, causing hypotension refractory to fluid recuscitation.

Lab/Physical Findings

- Physical exam findings—fever, tachypnea, warm skin and full peripheral pulses (from vasodilation), hypotension, poor urinary output.
- Positive blood cultures (negative about 50% of the time, particularly if drawn after antibiotics are started).

WARD TIP

Adequate (at minimum) urine output is 0.5 cc/kg/hr = 35 cc/hr for average 70-kg person.

The Continuum

SIRS → Sepsis → Severe Sepsis → Septic Shock

Septic shock is the most severe manifestation of infection in a continuum. Milder manifestations of infection are classified as systemic inflammatory response syndrome (SIRS), sepsis, and severe sepsis (Figure 5-1 and Table 5-1).

WARD TIP

Poor prognostic signs in septic shock:
- Disseminated intravascular coagulation (DIC).
- Multiple organ failure.

Treatment

- Fluids!!
- Source control
- Antibiotics: **Start broad-spectrum antibiotics early** and empirically treat until blood cultures come back!!
  - Surgical drainage of abscess or focus of infection.
- If blood pressure is unresponsive to fluids, use pressors, classically norepinephrine (Levophed).
- Tight glycemic control (maintain blood glucose < 180).

EXAM TIP

Gram-negative bacteria are notorious for causing septic shock.
Most common gram-negative organisms: Escherichia coli, Klebsiella, Pseudomonas aeruginosa.
Top three gram-positive organisms: Staphylococcus aureus, Enterococcus, coagulase-negative Staphylococcus.

anaPhylactic shock

Systemic type I hypersensitivity reaction causing chemically mediated angioedema and increased vascular permeability, resulting in hypotension and/or airway compromise.

WARD TIP

Even when SIRS criteria are met, infection is present < 50% of the time.

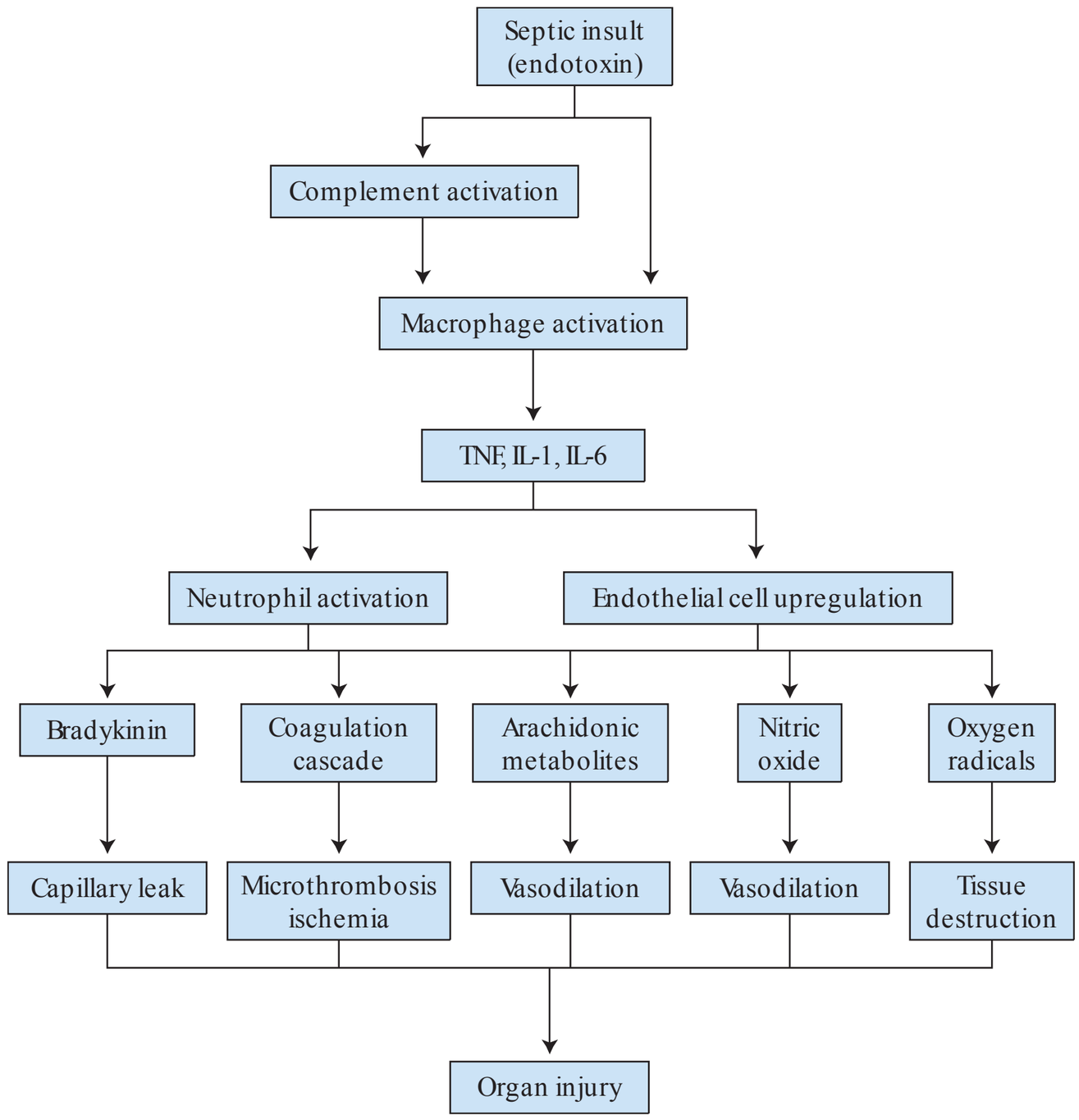


FIGURE 5-1. Schematic depiction of physiologic consequences of septic insult.

TABLE 5-1. Clinical Spectrum of infection and Systemic inflammatory response Syndrome (SIRS)

Term	Definition
Infection	Identifiable source of microbial insult
SIRS	Two or more of following criteria Temperature ≥ 38°C or ≤ 36°C Heart rate ≥ 90 beats/min Respiratory rate ≥ 20 breaths/min or $Paco_2$ ≤ 32 mmHg or mechanical ventilation White blood cell count ≥ 12,000/μL or ≤ 4,000/μL or ≥ 10% band forms
Sepsis	Identifiable source of infection + SIRS
Severe sepsis	Sepsis + organ dysfunction
Septic shock	Sepsis + cardiovascular collapse (requiring vasopressor support)

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 4.

Salient Physical Findings

- Urticaria and anigoedema (especially around lips)
- Laryngeal edema (stridor), wheezing

t reatment

- Epinephrine
- Antihistamines (diphenhydramine)
- Steroids

neuRogenic shock

Central nervous system (CNS) injury causing disruption of the sympathetic system, resulting in unopposed vagal outf ow and vasodilation. It is characterized by hypotension and bradycardia (absence of ref ex sympathetic tachycardia and vasoconstriction). Usually secondary to spinal cord injury of cervical or high thoracic region.

t reatment

- IV f uids: Usually patients respond; helps to place patient in Trendelenburg position.
- Vasopressors: Used early if patient is unresponsive to f uids.

caRDiogenic shock

Pump failure, resulting in decreased CO. This can be caused by myocardial infarction (MI), arrhythmias, valvular defects, heart failure, or cardiac contusion. Wedge pressure and SVR are elevated.

Findings

Patients will have cold, clammy skin from peripheral vasoconstriction. Additionally, they will have jugular venous distention (JVD), dyspnea, bilateral crackles, and S3/4 gallop. Chest x-ray (CXR) will show bilateral pulmonary congestion. Echocardiography demonstrates poorly contractile left ventricle, pulmonary capillary wedge pressure (PCWP) > 20 mmHg, cardiac index (CI) < 2.0.

t reatment

- Correct electrolyte abnormalities: Most commonly hypokalemia and hypomagnesemia.
- Pain control: IV morphine sulfate or fentanyl to minimize anxiety.
- Antiarrythmics, cardiac pacing, or cardioversion for pathologic dysrhythmias or heart block.
- Treatment of cardiogenic shock is specific to cause:
  - Acute MI: Oxygen, nitroglycerin, aspirin, IV morphine, cardiology consultation.
  - Cardiac contusion: Infusion of inotropes. Intra-aortic balloon pump can provide temporary treatment.
- **Left heart failure or biventricular failure:**
  - Isolated left ventricular failure: Pressors, and reduce afterload.
  - Congestive heart failure: Diuretics and vasodilators (nitrates) to decrease preload. May or may not use pressors.
  - Classically, the following inotropes can be used:
    - Dobutamine: Used after dopamine to increase cardiac contractility.
    - Amrinone and milrinone (phosphodiesterase inhibitors): Used in patients unresponsive to dopamine or dobutamine.
  - Intra-aortic balloon pump (IABP): Used as mechanical support in patients who do not respond to pressors or inotropes. IABP increases

WARD TIP

Causes of anaphylaxis: Drugs (penicillin), radiocontrast, insect bites (honeybee, fire ant, wasp), and food (shellfish, peanut butter).

EXAM TIP

Remember, type I hypersensitivity reactions are immunoglobulin E (IgE) mediated and require prior exposure.

EXAM TIP

Classic findings in spinal shock: Hypotension, bradycardia, and absence of rectal tone on digital rectal examination.

WARD TIP

MI is most common cause of cardiogenic shock.

Table 5-2. Types of Shock and Their Hemodynamic Profiles

	PCWP	CO	SVR
Hypovolemic shock	↓↓	↓	↑
Cardiogenic/obstructive shock	↑	↓↓	↑
Neurogenic shock	↓	↓	↓
Distributive shock	↓ or normal	↑	↓↓

CO, and decreases work of heart by reducing systolic afterload and increasing diastolic perfusion to coronary arteries. Device can be inserted percutaneously via femoral artery at the ICU bedside.

- **Isolated right heart failure:**
  - Most commonly caused by right ventricular infarction or PE.
  - Give fluids (maintains preload).

See Table 5-2.

Obstructive shock

Extracardiac causes of cardiac failure. They can be divided into pulmonary vascular etiologies (massive pulmonary embolism, severe pulmonary hypertension), or mechanical causes (tension pneumothorax, pericardial tamponade, constrictive pericarditis).

Findings

Patients will have cold clammy skin from peripheral vasoconstriction. They will have physical findings similar to patients with cardiogenic shock. CXR findings will vary depending on the reason for obstructive shock. Pulmonary embolism may not result in acute cardiopulmonary abnormalities. Pneumothorax will be visible on CXR. Pericardial effusion (if chronic) will present as an enlarged cardiac silhouette. Echocardiography would show pericardial effusions, and CT angiography of the chest will reveal the PEs Swan–Ganz catheter will be helpful to diagnose pulmonary hypertension.

Treatment

- Treatment of obstructive shock is specific to cause:
  - Pulmonary embolism: possible embolectomy, anticoagulation (heparin, Coumadin)
- Pulmonary hypertension: pulmonary vasodilators (sildenafil, remodulin), afterload reduction
- Cardiac tamponade: Percardiocentesis
- Constrictive pericarditis: NSAID, steroids

EXAM TIP

First physical exam and ECG finding of PE is tachycardia.
Classic ECG findings: $S_1Q_3T_3$, S wave in lead I, Q-wave and T-wave inversion in lead III. This is not sensitive or specific, however.

Swan–Ganz Catheter

The Swan–Ganz catheter (pulmonary artery catheter) is used with ICU and shock patients in order to obtain information relevant to fluid and volume status (it has never been shown to change mortality and its use is weaning). It is threaded via the subclavian or internal jugular into superior vena cava → right atrium → right ventricle → pulmonary artery.

The following are some of the measurements obtainable through the Swan–Ganz that will allow a better understanding of the different types of shock (Table 5-3):

- **Pulmonary artery occlusion pressure (PAOP or PCWP)** (normal 6–12 mmHg) This reflects the pressures of the left ventricle (end-diastolic pressure). It can be thought of as preload.
  Clinical context: If the pump fails, pressures in the left ventricle increase and you will have an increased wedge.
- **CO** (normal 4–8 mmHg) Remember, CO = SV × HR. The Swan–Ganz allows CO to be measured via the **thermodilutional technique:** The temperature change is measured at the distal end of the catheter when cold fluid is injected from the proximal port. The difference in temperature reflects the CO, which can also be thought of as pump function.
- **Clinical context:** If you have an MI and lose wall motion, your SV will be decreased. Likewise, if you hemorrhage and have no preload, your SV will decrease as well.
- **SVR** (usually divided by body surface area to give systemic vascular resistance index [SVRI]): SVR reflects the vascular resistance across the systemic circulation (it can be thought of as afterload as well).
- **Clinical context:** Distributive shock causes vessels to dilate and leak, causing SVR to decrease. Cardiogenic, obstructive, and hypovolemic shock results in vasoconstriction, causing SVR to increase.

WARD TIP

End points of resuscitation for shock of any etiology:

- Normalization of lactate (marker of oxygen debt), base deficit, and pH.
- Normalization of mixed venous oxygen saturation (marker of oxygen delivery and extraction) and CO.
- Urine output—most useful measure of effective fluid resuscitation (marker of renal perfusion).

WARD TIP

Risks of the Swan–Ganz:

- Infection.
- Arrhythmia (the guidewire can irritate the endocardium and trigger this). Injury of the pulmonary artery.

TABLE 5-3. Normal Ranges for Selected Hemodynamic Parameters in Adults

Parameter	Normal Range
CVP	0–6 mmHg
Right ventricular systolic pressure	20–30 mmHg
Right ventricular diastolic pressure	0–6 mmHg
PAOP	6–12 mmHg
Systolic arterial pressure	100–130 mmHg
Diastolic arterial pressure	60–90 mmHg
MAP	75–100 mmHg
Q_T	4–6 L/min
Q_{T^*}	2.5–3.5 $L \cdot min^{-1} \cdot m^{-2}$
SV	40–80 mL
SVR	800–1,400 dyne · sec · cm^{-5}
SVRI	1,500–2,400 dyne · sec · $cm^{-5} \cdot m^{-2}$
PVR	100–150 dyne · sec · cm^{-5}
PVRI	200–400 dyne · sec · $cm^{-5} \cdot m^{-2}$
Ca_{O_2}	16–22 mL/dL
Cv_{O_2}	~15 mL/dL blood
D_{O_2}	400–660 $mL \cdot min^{-1} \cdot m^{-2}$
V_{O_2}	115–165 $mL \cdot min^{-1} \cdot m^{-2}$

Ca_{O_2} = arterial oxygen content; Cv_{O_2} = central venous oxygen pressure; CVP = mean central venous pressure; D_{O_2} = systemic oxygen delivery; MAP = mean arterial pressure; PAOP = pulmonary artery occlusion (wedge) pressure; PVR = pulmonary vascular resistance; PVRI = pulmonary vascular resistance index; Q_T = cardiac output; Q_{T^*} = cardiac output indexed to body surface area (cardiac index); SV = stroke volume; SVI = stroke volume index; SVR = systemic vascular resistance; SVRI = systemic vascular resistance index; V_{O_2} = systemic oxygen utilization.

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 366.

WARD TIP

How is the wedge pressure obtained? The "wedge" pressure is obtained by inflating the distal end of the Swan–Ganz and "floating" this "balloon" down the pulmonary artery until it "wedges." The pressure measured equals the pulmonary capillary pressure, and this, theoretically, represents the left atrial pressure and ultimately the left ventricular end-diastolic pressure (LVEDP).

WARD TIP

What is the significance of the wedge pressure? It reflects the left ventricular pressure, which will be increased with left ventricular failure.

Pressors and Inotropes

A group of vasoactive drugs that are the final line of defense in treating shock (Table 5-4).

Effects and Side Effects

Generally, pressors are used to increase CO or SVR. All of them have important side effects that can limit their use.

These side effects are easily predicted based on the drug's action. For example, in addition to stimulating β_1 receptors, dobutamine stimulates β_2 (which causes vasodilation). The β_2 stimulation causes the side effect of hypotension.

Furthermore, remember that virtually any direct stimulation of the heart (β_1) can cause the side effect of arrhythmias.

Dobutamine

- **Action:** Strong stimulation β_1 receptors (ionotropic/chronotropic effects on the heart) with a mild stimulation of β_2 (vasodilation).
- **Result:** ↑ CO, ↓ SVR.
- **Typical use:** Cardiogenic shock.

Isoproterenol

Similar to dobutamine.

- **Action:** Strong stimulation of β_1 receptors (ionotropic/chronotropic effects on the heart) and β_2 (vasodilation).

TABLE 5-4. Relative Selectivity of Adrenoceptor Agonists

	Relative Receptor Affinities
Alpha agonists	
Phenylephrine, methoxamine	$\alpha_1 > \alpha_2 >>>>> \beta$
Clonidine, methylnorepinephrine	$\alpha_2 > \alpha_1 >>>>> \beta$
Mixed alpha and beta agonists	
Norepinephrine	$\alpha_1 = \alpha_2; \beta_1 >> \beta_2$
Epinephrine	$\alpha_1 = \alpha_2; \beta_1 = \beta_2$
Beta agonists	
Dobutamine	$\beta_1 > \beta_2 >>>> \alpha$
Isoproterenol	$\beta_1 = \beta_2 >>>> \alpha$
Terbutaline, metaproterenol, albuterol, ritodrine	$\beta_2 >> \beta_1 >>>> \alpha$
Dopamine agonists	
Dopamine	$D_1 = D_2 >> \beta >> \alpha$
Fenoldopam	$D_1 >> D_2$

Reproduced, with permission, from Katzung BG. Basic & Clinical Pharmacology. 10th ed. New York: McGraw-Hill; 2007: 124.

- **Result:** ↑ CO, ↓ SVR.
- **Typical use:** Cardiogenic shock with bradycardia.

milRinone

Milrinone is technically not a pressor, but it is an important inotrope used in the ICU.

- **Action:** Phosphodiesterase inhibitor, which results in increased cyclic adenosine monophosphate (cAMP). This has positive ionotropic effects on the heart and also vasodilates.
- **Result:** ↑ CO, ↓ SVR.
- **Typical use:** Heart failure/cardiogenic shock.

DoPamine

Dopamine has different action depending on the dose.

low Dose (1–3 µg/kg/min): "renal Dose"

- **Action:** Stimulation of dopamine receptors (dilates renal vasculature) and mild β_1 stimulation.
- **Typical use:** None.

intermediate Dose (5–10 µg/kg/min): "Cardiac Dose"

- **Action:** Stimulation of dopamine receptors, moderate stimulation of β_1 receptors (heart ionotropy/chronotropy), and mild stimulation of α_1 receptors (vasoconstriction).
- **Result:** ↑ CO.
- **Typical use:** Cardiogenic shock.

High Dose (10–20 µg/kg/min)

- **Action:** Stimulates dopamine receptors, β_1 receptors (heart ionotropy/chronotropy), and strong stimulation of α_1 receptors (vasoconstriction).
- **Result:** ↑↑ SVR.
- **Typical use:** Septic shock (replaced by norepinephrine).

WARD TIP

The concept of low-dose dopamine's being a "renal dose" and helping perfuse the kidney has been debunked. It dilates the vasculature, but no evidence shows that it is renal protective or improves renal failure.

noRePinePhRine

- **Action:** Strong stimulation of α_1 receptors (vasoconstriction), moderate stimulation of β_1 receptors (heart ionotropy/chronotropy).
- **Result:** ↑↑ SVR, ↑ CO.
- **Typical use:** Septic shock.

ePinePhRine

- **Action:** Strong stimulation of β_1 and β_2 receptors. Also $\alpha_{1,2}$ stimulation (vasoconstriction).
- **Result:** ↑↑ SVR, + / - ↑ CO, bronchodilation.
- **Typical use:** Anaphylaxis, septic shock, cardiopulmonary arrest.

EXAM TIP

Norepinephrine, followed by vasopressin, are the pressors of choice in septic shock.

EXAM TIP

- When resuscitating, monitor urine output (minimum 1 mL/kg/hr) and CVP (no greater than 15 mmHg).

WARD TIP

Pressure support is an important weaning mode off the ventilator; it provides enough positive pressure to keep alveoli open but breaths (ventilation) are triggered by patient.

EXAM TIP

Complications of mechanical ventilation:

- Ventilator-associated pneumonia.
- Barotrauma and tension pneumothorax.
- Decreased venous return (preload) and CO.

WARD TIP

Strategies to improve oxygenation:

- Increase FiO_2.
- Increase positive end-expiratory pressure (PEEP).

WARD TIP

PEEP is used primarily in congestive heart failure (CHF) or acute respiratory distress syndrome (ARDS). It maintains alveoli open, allowing more time for gas exchange. It is therefore used to increase the oxygen level. Problems with PEEP include hypotension (decreases preload).

Phenylephrine

- **Action:** Strong stimulation of α_1 receptors (vasoconstriction).
- **Result:** ↑↑ SVR.
- **Typical use:** Septic shock, neurogenic shock, anesthesia-induced hypotension.

Mechanical Ventilation

The goals of mechanical ventilation are to:

1. Improve gas exchange.
2. Decrease the work of breathing.

Indications for intubation:

- Airway protection (patients with GCS < 8).
- Failure to oxygenate—hypoxia despite high oxygen delivery content and clinical signs of respiratory distress (excess work of breathing).
- Failure to ventilate leading to progressive hypercapnia with acidosis and signs of mental status change.

setting the ventilator

See Table 5-5.

Several parameters have to be set for the ventilator. You need to define the mode, respiratory rate, tidal volume, and the FiO_2.

- **Mode:** Choose assist control (AC), synchronized intermittent mandatory ventilation (SIMV), pressure support (PS), or continuous positive airway pressure (CPAP).
- **Respiratory rate** (for AC or SIMV only): Usually 10–20.
- **Tidal volume** (for AC or SIMV only): Usually 400–600 cc (6–8 cc/kg).
- **FiO_2:** Always start at 100% and titrate down, maintaining the pulse oximetry > 90%. Keep FiO_2 below 60% to minimize oxygen-induced free radical injury.

example

Initial AC mode setting used in the surgical patient: AC 12–16, tidal volume 6–8 cc/kg (ideal body weight), FiO_2 = 100% (reduced after initial ABG), PEEP 5.

adjusting the ventilator

1. Based on ABG (interpretation discussed in previous chapter).
2. Minute ventilation.

$$\text{Minute Ventilation} = \text{Respiratory Rate} \times \text{Tidal Volume}$$

You can therefore adjust either the rate or the tidal volume to change the minute ventilation.

Know this simple rule: Increasing minute ventilation will decrease Pco_2 and increase pH. Decreasing minute ventilation will increase Pco_2 and decrease pH.

Oxygenation

FiO_2 and PEEP are the ventilator parameters that adjust oxygenation.

Table 5-5. Various Modes of Mechanical Ventilation

Continuous positive airway pressure (CPAP)	Noninvasive ventilation that applies constant positive pressure, with no variation in breathing cycle. Patient must breathe on his or her own. CPAP keeps inspiratory airway pressure above atmospheric pressure without increasing work of breathing; improves functional residual capacity and compliance. Can be used to help avoid intubation. Mode of choice for sleep apnea.
Synchronized intermittent mandatory ventilation (SIMV)	Patient breathes on his or her own, plus receives a preset rate of MV that is synchronized to and delivered with the patient's breath. Pressure support is often added to spontaneous breathing (gives patient initial boost of pressure to overcome airway resistance).
Assist-control ventilation (ACV) (may be volume controlled or pressure controlled)	Each breath initiated by patient triggers machine to deliver the set tidal volume (volume control) or set peak inspiratory pressure (pressure control). A set tidal volume/pressure is given a set number of times per minute, even if patient is breathing less than the preset respiratory rate.
Pressure support ventilation (PSV)	Each breath initiated by patient triggers the machine to deliver an initial "boost of pressure" with variable flow of air into lungs. The patient determines the rate, duration of inspiration, and tidal volume. This boost helps the patient overcome resistance of the endotracheal tube and reduces work of breathing. This mode can be used alone, as the only ventilator setting, or in conjunction with the IMV/SIMV modes. Pressure support is typically set at between 5 and 20. This mode is used in weaning of ventilator.

Acute Respiratory Distress Syndrome (ARDS)

Acute lung injury due to inflammatory process in both lungs causing increased permeability of the capillaries and severe ventilation/perfusion mismatch. ARDS is a disease of altered lung compliance. These patients are tachypneic and hypoxic and have bilateral crackles on lung exam.

The diagnostic criteria are:

1. Bilateral, fluffy infiltrates on CXR
2. PaO_2/FiO_2 ratio < 200.
3. No evidence of heart failure (PCWP ≤ 18 mmHg).
4. Acute onset.
5. Presence of an underlying cause.

EXAM TIP

What are the causes of failure to wean off the ventilator?

1. Anxiety/agitation.
2. Drugs (usually sedatives).
3. Electrolyte abnormalities (hypophosphatemia).
4. Diaphragm dysfunction (neuromuscular dysfunction).
5. Hypothyroidism/malnutrition.
6. Excess CO_2 production (overfeeding).

WARD TIP

Pco_2 is the marker of ventilation.
Po_2 is the marker of oxygenation.

WARD TIP

Try to titrate down the FiO_2 (< 50% is ideal) in order to avoid oxygen toxicity. Oxygen toxicity is thought to be caused by oxygen-free radicals damaging the lung interstitium.

WARD TIP

The CXR does not reliably distinguish ARDS from CHF. A Swan–Ganz catheter can be useful in this matter.

- PCWP < 18 = ARDS
- PCWP > 18 = CHF

causes

There is a wide variety of causes of ARDS. Common causes include:

- Direct lung injury:
  - Pneumonia
  - Aspiration
  - Near drowning
- Indirect causes:
  - Sepsis (most common of all causes).
  - Massive transfusion.
  - Severe trauma, burns, toxins.
  - Pancreatitis.

management

1. It is necessary to intubate because of hypoxemia.
2. Treat underlying cause, especially infections.
3. **Low tidal volume ventilation (6–8 cc/kg).**
4. PEEP is often used to improve gas exchange and keep lungs open at relatively low lung volumes.
5. FiO_2 is kept at $\leq 60\%$ to avoid free radical injury.

Fluids, Electrolytes, and nutrition

Anatomy of Body Fluids 74
- Total Body Water 74
- Intracellular Fluid (ICF) 74
- Extracellular Fluid (ECF) 74

Normal Fluid and Electrolyte Exchange 74
- Water Movement Between ICF and ECF 74

Renal Control of Fluids/Electrolytes 76
- Volume Deficit (Dehydration) 76

Volume Excess 78
- Causes 78
- Signs and Symptoms 78
- Treatment 78

Ongoing Fluid Loss 79
- Causes 79

Assessing Volume Status 79
- Vital Signs 79
- History and Physical Exam in Hypervolemia 79
- History and Physical Exam in Hypovolemia 79
- Input, Output, Weight 80
- Urine Output (UO) 80
- Lab 80

Sodium Balance 80
- Hyponatremia 80
- Hypernatremia 81

Potassium Balance 83
- Hypokalemia 83
- Hyperkalemia 84

Calcium Balance 85
- Hypocalcemia 86
- Hypercalcemia 87

Indications for Nutritional Support 87
- Enteral 87
- Total Parenteral Nutrition (TPN) 87

Nutrient Requirements 88
- Caloric Requirements 88
- Respiratory Quotient (RQ) of Individual Substrates 88
- Protein Requirement 88

Enteral Nutrition 88
- Complications 89

Acid–Base Disorders 89
- Metabolic Acidosis 89
- Metabolic Alkalosis 90
- Respiratory Acidosis 90
- Respiratory Alkalosis 90

EXAM TIP

normal Plasma osmolality 285–295 mmol/l

Calculated Plasma osmolality (POSMc) = 2[Na] + [glucose]/18 + [BUN]/2.8

Where [Na] is in mmol/L and [BUN], [glucose] in mg/dL

Osmolar Gap = measured osmolality − POSMc

Normal Osmolar Gap < 10 mmol/L

Osmolar Gap > 10 mmol/L—think lactic acid, ketones, methanol, ethanol.

WARD TIP

Even without intake, you must excrete 800 mL/day in urine waste products.

WARD TIP

Insensible loss increases with fever and hyperventilation.

WARD TIP

May lose 1,500 mL/day with an unhumidified tracheostomy and hyperventilation.

Anatomy of Body Fluids

Total Body Water

- Fifty to 70% of total body weight.
- Greater in lean individuals because fat contains little water, average 60%.
- Greatest percentage in newborns 70%, then decreases with age to around 50%.
  - **Example:** Average 70-kg male would be 42 L water since 1 L of water = 1 kg.
- Made up of two compartments—ICF and ECF.

Intracellular Fluid (ICF)

- Mostly in skeletal muscle mass, thus slightly lower in females (50%) than males (60%).
- Cell wall separates the ICF from the ECF and acts as a semipermeable membrane.

extracellular Fluid (ecF)

- Made up of plasma and interstitial (extravascular) fluid.
- Capillary membrane separates plasma and interstitial fluid and acts as a semipermeable membrane.

	Intracellular Compartment	Extracellular Compartment	
	⅔ = 67% TBW	⅓ = 33% TBW	
		Interstitial	Plasma
		¾ ECC = 25% TBW	¼ ECC = 8% TBW
In 70-kg man (42 L TBW) = 28 L		11 L	3 L
In 45-kg woman (22.5 L TBW) = 15 L		5.6 L	1.8 L
	↑ Cell membrane		↑ Capillary wall

Normal Fluid and Electrolyte Exchange

Water Movement Between ICF and ecF

Water flows freely between the three compartments, shifting compartments to maintain osmotic equilibrium between them (Figure 6-1). See Table 6-1.

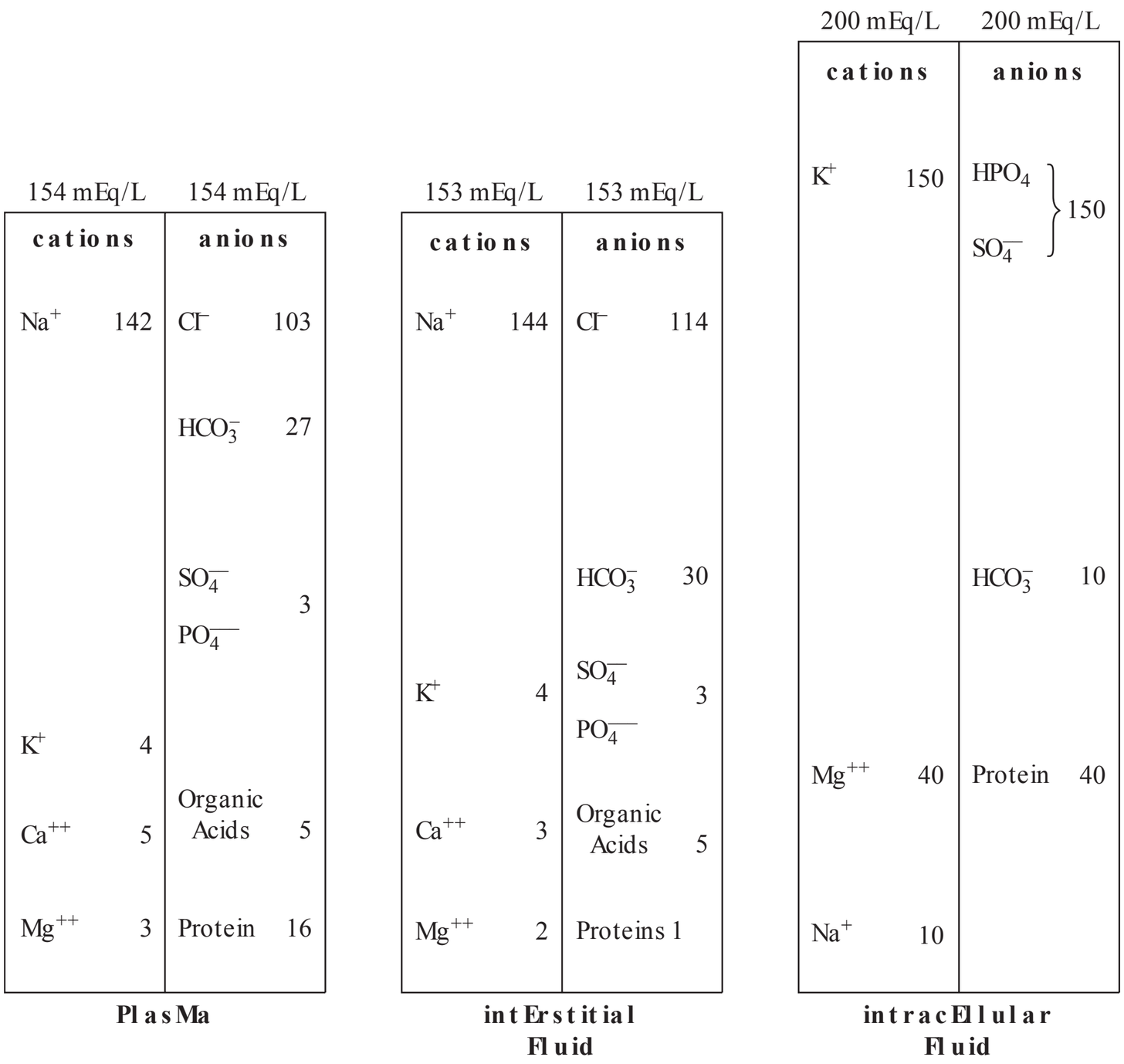


FIGURE 6-1. chemical composition of body fluid compartments. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 45.)

TABLE 6-1. signs and symptoms of Volume disturbances

System	Volume Deficit	Volume excess
Generalized	Weight loss	Weight gain
	Decreased skin turgor	Peripheral edema
Cardiac	Tachycardia	Increased cardiac output
	Orthostasis/hypotension	Increased central venous pressure
	Collapsed neck veins	Distended neck veins
		Murmur
Renal	Oliguria	
	Azotemia	
Gastrointestinal	Ileus	Bowel edema
Pulmonary		Pulmonary edema

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 45.

Renal Control of Fluids/Electrolytes

See Figure 6-2.

- Distal tubules—reabsorption of Na in exchange for K and H secretion.
- Affected by adrenocorticotropic hormone (ACTH) and aldosterone.
- Aldosterone directly stimulates K secretion and Na reabsorption from the distal tubule.

VOLUME DEFICIT (DEHYDRATION)

Most common fluid disorder.

causes

Losses that Mimic ECF

- Hemorrhage.
- Loss of gastrointestinal (GI) fluid—vomiting, nasogastric (NG) suction, diarrhea, fstular drainage.
- Postoperative fluid sequestration (third spacing): Intestinal obstruction.
- Intra-abdominal and retroperitoneal inflammation (e.g., pancreatitis, peritonitis).
- Systemic inflammatory response syndrome (SIRS), burns, sepsis, pancreatitis.

Losses that Are Principally Water

- Fever.
- Osmotic diuresis.
- Diabetes insipidus.
- Prolonged water deprivation.
- Inadequate input during procedure.

signs and symptoms

See Table 6-1.

- Central nervous system (CNS) and cardiovascular (CV) signs occur early with acute loss.
- CV signs are secondary to a decrease in plasma volume.
- Tissue signs may be absent until the deficit has existed for 24 hours.

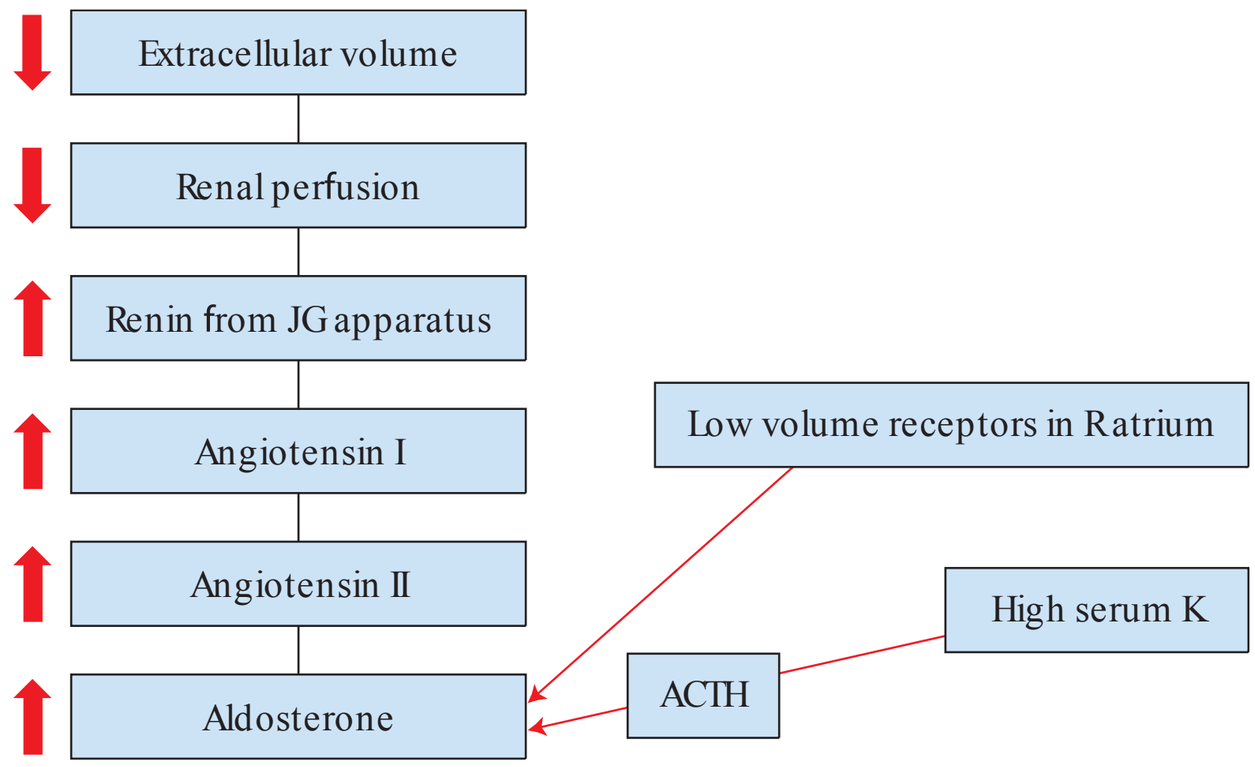


FIGURE 6-2. renal mechanism of fluid and electrolyte balance.

- Tissue signs may be difficult to assess in the elderly patient or patient with recent weight loss.
- Body temperature varies with environment—cool room may mask fever.
- After partial correction of volume deficit, the temperature will generally rise to the appropriate level.
- Severe volume depletion depresses all body systems and interferes with the clinical evaluation of the patient.
- Volume depleted patient with severe sepsis from peritonitis may be afebrile and have normal white blood count (WBC), complain of little pain, and have unremarkable findings on abdominal exam. This may change dramatically when the ECF is restored.
- History items important for evaluating fluid deficits include:
  - Weight change, intake (quantity and composition), output, general medical status.
  - Degree of dehydration dependent on acute loss of body weight and is assessed clinically:
    - Mild—3% for adults, 5% for kids
    - Moderate—6% for adults, 10% for kids
    - Severe—9% for adults, 15% for kids

treatment

- Goal is to replace this deficit in most patients over the next 24 hours.
- The amount of fluid the patient is missing needs to be combined with the expected maintenance fluid for the next 24 hours.
- Rehydration is done over this period of time to try to allow continual equilibration between the reexpanded intravascular space and the contracted ECF and ICF.
- The initial intervention is to give a large amount of fluid as a volume expander; 20 mL/kg of normal saline (NS) or Ringer's lactate (LR) is given as a bolus. During the remaining 8 hours, the expected maintenance fluid is given plus about half of the remaining calculated loss. Over the remaining 16 hours, the other half of the remaining calculated loss is given along with the maintenance fluid.
- Volume expansion can be accomplished with crystalloid (NaCl, LR, etc.) or colloid (albumin, blood products).

Crystalloid

- Dextrose solutions are used to deliver free water to the body (dextrose is quickly metabolized).
- 0.9% NaCl quickly adds volume to the intravascular space.
- Goal is to expand the intravascular space.

Colloids

- Include packed RBCs, FFP, albumin.
- Stay mainly within intravascular space if the capillary membranes are intact.
- Expensive.
- Indications:
  - Hypovolemic patients with excess Na and water—such as ascites, congestive heart failure (CHF), postcardiac bypass patients.
  - Patients unable to synthesize enough albumin or other proteins to exert enough oncotic pressure—such as liver disease, transplant recipients, resections, malnutrition.
  - Severe hemorrhage or coagulopathy—packed red blood cells (PRBCs) and fresh frozen plasma (FFP) may increase hematocrit to help correct coagulopathy.



WARD TIP

When replacing fluids, remember that:

- Large volumes may lead to peripheral and/or pulmonary edema.
- Large amounts of dextrose may cause hyperglycemia.
- Large amounts of NS may cause hyperchloremic metabolic acidosis.
- Ringer's lactate given when patient is hypovolemic and in metabolic alkalosis (i.e., from NG tube, vomiting) may worsen the alkalosis when the lactate is metabolized.

Volume Excess

causes

Isotonic

- Iatrogenic—intravascular overload of IV fluids with electrolytes.
- Increased ECF without equilibration with ICF—especially postoperative or trauma when the hormonal responses to stress are to decrease Na and water excretion by kidney.
- Often secondary to renal insuff ciency, cirrhosis, or CHF.

Hypotonic

- Inappropriate NaCl-poor solution as a replacement for GI losses (most common).
- Third spacing.
- Increased antidiuretic hormone (ADH) with surgical stress, inappropriate ADH (SIADH).

EXAM TIP

Third spacing is the shift of ECF from the plasma compartment to elsewhere, such as the interstitial or transcellular spaces.

Hypertonic

Most common cause: excessive Na load without adequate water intake:

- Water moves out of the cells because of increased ECF osmolarity.
- Causes an increase in intravascular and interstitial fluid.
- Worse when renal tubular excretion of water and/or Na is poor.
- Can also be caused by rapid infusion of nonelectrolyte osmotically active solutes such as glucose and mannitol.

sIgns and syMpToMs

See Table 6-2.

Tr eaTMenT

- Restriction of Na and fluids for isotonic hypervolemia.
- Free water replacement for hypertonic hypervolemia.
- Saline for hypotonic hypervolemia.

TABLE 6-2. Composition of Gastrointestinal Secretions

Type of Secretion	Volume (ml/24 h)	Na (meq/l)	K (meq/l)	Cl (meq/l)	HCO_3^- (meq/l)
Stomach	1,000–2,000	60–90	10–30	100–130	0
Small intestine	2,000–3,000	120–140	5–10	90–120	30–40
Colon		60	30	40	0
Pancreas	600–800	135–145	5–10	70–90	95–115
Bile	300–800	135–145	5–10	90–110	30–40

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 46.

- Diuresis with furosemide 10–50 mg:
  - Be sure to replace K as needed.
  - Be careful not to over diurese—must maintain kidney and brain perfusion as well as appropriate cardiac output.

Ongoing Fluid Loss

- **Rule of thumb:** Replace one half of the "usual" ongoing losses along with the assumed maintenance and the rehydration replacement fluid.
- Electrolyte content of the ongoing loss can be either assumed based on serum electrolyte values or can be determined by direct electrolyte measurement of the fluid (see Table 6-2).

Causes

- Fever: Each °C above 37°C adds 2.0–2.5 mL/kg/day of insensible water loss.
- Loss of body fluids: From vomit, NG suction, fistulas.
- Third-space losses:
  - Adults—approximately 1 L of third-space fluid intra-abdominally for each quadrant of the abdomen that is traumatized, inflamed, or operated on.
  - Kids—approximately one fourth of calculated maintenance fluid per 24-hour period is sequestered for each quadrant of the abdomen that is traumatized, inflamed, or operated on.
- Burns:
  - See Burns chapter for estimating volume losses and replacement.
  - Osmotic diuresis:
    - Secondary to urea, mannitol, or glucose.
    - Urine electrolytes should be checked to determine the appropriate replacement fluid, if one is necessary.

WARD TIP

The average (70-kg) adult patient needs about 2.5 L/day (1,000 + 500 (20 × 50) = 2,500) or 100 mL/hr (2,500/24 = 104) unless other factors warrant a higher rate.

EXAM TIP

Calculating free water deficit (FWD):

- FWD = Normal Body Water (NBW) − Current Body Water (CBW)
- NBW = 0.6 × body weight in kg
- CBW = (NBW) [normal serum Na/measured serum Na]

Assessing Volume Status

Vital signs

- Early signs of hypovolemia: Tachycardia, decreased pulse pressure, orthostatic blood pressure (BP).
- BP is not persistently lowered until 20–30% of circulating volume is lost.

History and PHysiCal exam in HyPer Volemia

- **Hx:** Weight gain, recent myocardial infarction (MI), shortness of breath, orthopnea.
- **PE:** Jugular venous distention (JVD), rales, S3, pitting edema, ascites.

History and PHysiCal exam in HyPo Volemia

- **Hx:** Weight loss, vomiting, diarrhea, burns.
- **PE:** Flat neck veins, poor tissue turgor, dry mucous membranes, cool extremities, slow capillary refill.

Input, Output, Weight

Daily weight is one of the best methods for assessing volume status.

Urine Output (UO)

- Normal UO: 0.5 cc/kg/hr for adults, 1 cc/kg/hr for kids.
- Low UO: Hypovolemia, renal failure, low flow states.
- High UO: Hypervolemia, diabetes insipidus, osmotic diuresis, post-obstructive diuresis.

Lab

- Check daily serum electrolytes in intensive care unit (ICU) patients.
- Blood urea nitrogen (BUN)/creatinine (Cr) > 20 and FeNa < 1% indicates hypovolemia.
- BUN/Cr < 15 indicates adequate hydration.

Sodium Balance

Alterations in sodium balance cause fluid shifts between compartments. The clinical picture is most often neurologic, due to fluid shifts within the brain.

Hyponatremia

Definition

- < 130 mEq/L.
- First steps in hyponatremia: Determine volume status clinically, then determine plasma osmolality!

Step 1: Determine Plasma Osmolality

- Normal osmolality—pseudohyponatremia: Lab artifact due to increased lipids or plasma proteins → next step; check lipid profile or possible multiple myeloma.
- High osmolality—pseudohyponatremia: Due to increase of osmotically active molecules—glucose or mannitol.
- Low osmolality—true hyponatremia.

Step 2: Assess Volume Status

- Hypovolemia
- Euvolemia
- Hypervolemia

See below for discussion of each.

Hyponatremia with High Plasma Osmolality (Pseudohyponatremia)

Causes

Hyperglycemia, either physiologic or due to rapid infusion of glucose or mannitol will cause increased osmotic pressure that shifts fluid from the ICF to the ECF. The total body sodium in this case is normal but has become diluted due to the fluid shift.

The expected Na concentration can be calculated as follows: For every 100 mg/dL that glucose is increased over 100 mg/dL, the Na concentration falls 1.6 mEq/L. Remember "sweet 16."

WARD TIP

Chronic hyponatremia may be asymptomatic until < 120 mEq/L.

For every 100-mg/dL increment in plasma glucose above normal, the plasma sodium should decrease by 1.6 mEq/L.

For example, a patient with a glucose concentration of 500 mg/dL is expected to have a hyponatremia of around 133.6 mEq/L (4 × 1.6 = 6.4, 140 − 6.4 = 133.6).

Hyponatremia with Hypotonicity (true Hyponatremia)

True hyponatremia reflects excess ingestion of water that overwhelms the kidneys (either normal or diseased) or due to increased ADH. Hyponatremia is not due to increased excretion of sodium.

Hypovolemia (dehydration)

- Renal cause: Diuretics.
- Extrarenal cause: Vomiting, diarrhea, burns, pancreatitis.
- Differentiate using urine Na: Urine Na < 20 mEq/L indicates expected renal retention in the face of hypovolemia, suspect an extrarenal cause. Urine Na > 20 mEq/L indicates a renal cause.

Hypervolemia

- May be from CHF, cirrhosis, or nephrotic syndrome.
- Increased thirst and vasopressin.
- Edematous state.

Euvolemia

- SIADH: Most common cause of normovolemic hyponatremia.
- Increased vasopressin release from posterior pituitary or ectopic source causes decreased renal free water excretion.
- **Signs and symptoms:**
  - Hypo-osmotic hyponatremia (hyponatremia with hypotonicity).
  - Inappropriately concentrated urine (urine osmolality > 100 mOsm/kg).
  - Normal renal, adrenal, and thyroid function.
- **Causes:**
  - Neuropsychiatric disorders, malignancies (especially lung), and head trauma.
  - Glucocorticoid deficiency (Addison's disease)—cortisol deficiency causes hypersecretion of vasopressin.
  - Hypothyroidism—causes decreased CO and glomerular filtration rate (GFR), which leads to increased vasopressin secretion.
  - Primary polydipsia—usually seen in psychiatric patients who compulsively drink massive volumes of water.

signs and symptoms of (true) Hyponatremia

- **Signs:** Decreased reflexes, respiratory depression, seizures, coma (see Table 6-3).
- **Symptoms:** Nausea/vomiting, headache, lethargy, muscle cramps.
- Hypovolemic hyponatremia: Give 0.9% NaCl. Na repletion with saline isotonic to the patient, in order to avoid rapid changes in ICF volume. Major complication from rapid correction of chronic hyponatremia is **central pontine myelinolysis**.
- Hypervolemic hyponatremia: Correct underlying disorder—CHF, liver or renal failure.
- Euvolemic hyponatremia: Raise plasma Na (lower ICF volume)—restrict water intake.

EXAM TIP

- If hyponatremia is severely symptomatic (seizures), consider hypertonic (3%) saline.

EXAM TIP

- Hyponatremic Hypervolemia: correct with salt and water restriction, then furosemide (with potassium as needed), and ADH receptor antagonists (conivaptan).

hypernatremia

definition

> 145 mEq/L.

TABLE 6-3. Clinical Manifestations of Abnormalities in Serum Sodium

Body System	Hyponatremia
Central nervous system	Headache, confusion, hyper- or hypoactive deep tendon reflexes, seizures, coma, increased intracranial pressure
Musculoskeletal	Weakness, fatigue, muscle cramps/twitching
Gastrointestinal	Anorexia, nausea, vomiting, watery diarrhea
Cardiovascular	Hypertension and bradycardia if significant increases in intracranial pressure
Tissue	Lacrimation, salivation
Renal	Oliguria
Body System	**Hypernatremia**
Central nervous system	Restlessness, lethargy, ataxia, irritability, tonic spasms, delirium, seizures, coma
Musculoskeletal	Weakness
Cardiovascular	Tachycardia, hypotension, syncope
Tissue	Dry, sticky mucous membranes; red, swollen tongue; decreased saliva and tears
Renal	Oliguria
Metabolic	Fever

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 48.

Hypernatremia is always associated with hyperosmolarity. (Note that in the plasma osmolality equation, Na is the major factor).

causes

- Loss of water (dehydration!): Diabetes insipidus, diuretics, sweating, GI loss, burns, fistulas.
- Gain of sodium due to excess mineralocorticoid activity: Primary hyperaldosteronism, Cushing's, renal artery stenosis (hyperreninism), congenital adrenal hyperplasia (will cause concomitant hypokalemia).
- If thirst mechanism is intact and water is available, hypernatremia will not persist. Suspect hypernatremia in the young, elderly, and patients with altered mental status who may not have access to water.

symptoms

- Thirst.
- Restlessness, weakness, delirium.
- Hypotension and tachycardia.
- Decreased saliva and tears.
- Red, swollen tongue.
- Oliguria.

treatment

- Calculate FWD.
- If euvolemic: Replace water deficit with D_5W.
- If hypovolemic: Use NS. Correct one half of water deficit in first 24 hours; remaining water deficit over next 1–2 days.
- After euvolemic, slow D5W infusion to correct hypernatremia at rate between 8 and 12 mEq/dL/24 hours. Any faster than 12 mEq/dL/day, and there is risk of **cerebral edema**.

Potassium Balance

- Ninety-nine percent of K is in ICF. Therefore, small alterations in extracellular K balance can have significant clinical effects, particularly impaired electrical signaling in the heart, muscle, and nerve (see Table 6-4). Proper proportions of K^+ and Ca^+ must exist for their exchange across membrane channels that allow electrical conduction to occur.
- Cells act as a rapid potassium buffer. Kidney regulates long-term potassium control.

hypokaleMIa

definition

< 3.5 mEq/L.

TABLE 6-4. Clinical Manifestations of Abnormalities in Potassium, Magnesium, and Calcium

	Increased Serum Levels		
System	**Potassium**	**Magnesium**	**Calcium**
Gastrointestinal	Nausea/vomiting, colic, diarrhea	Nausea/vomiting	Anorexia, nausea/vomiting, abdominal pain
Neuromuscular	Weakness, paralysis, respiratory failure	Weakness, lethargy, decreased reflexes	Weakness, confusion, coma, bone pain
Cardiovascular	Arrhythmia, arrest	Hypotension, arrest	Hypertension, arrhythmia
Renal			Polyuria, polydipsia
	Decreased Serum Levels		
System	**Potassium**	**Magnesium**	**Calcium**
Gastrointestinal	Ileus, constipation		
Neuromuscular	Decreased reflexes, fatigue, weakness, paralysis	Hyperactive reflexes, muscle tremors, tetany, seizures	Hyperactive reflexes, paresthesias, carpopedal spasm, seizures
Cardiovascular	Arrest	Arrhythmia	Heart failure

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 49.

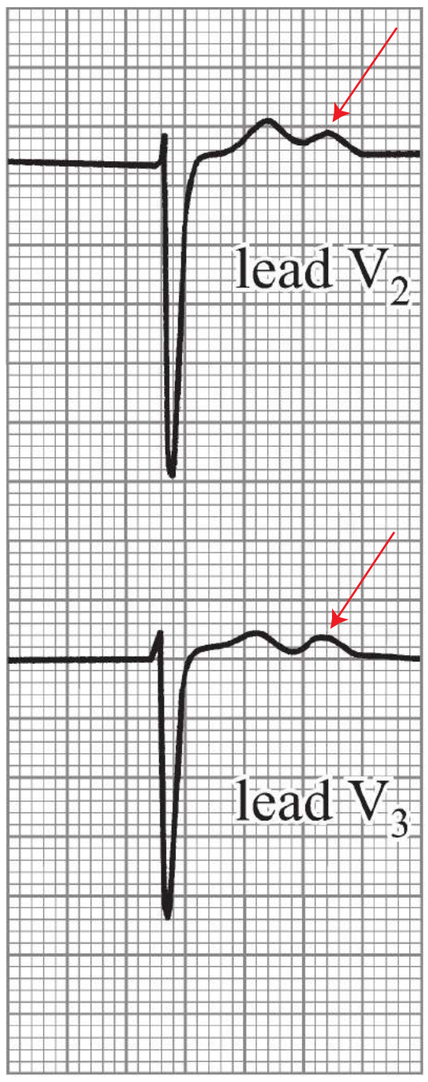


FIGURE 6-3. ECG demonstrating U wave of hypokalemia.

causes

- Most commonly due to excessive renal secretion.
- Loss of potassium due to excess mineralocorticoid activity: Primary hyperaldosteronism, Cushing's, renal artery stenosis (hyperreninism), congenital adrenal hyperplasia (will cause concomitant hypernatremia).
- Movement of K into cells due to insulin, catecholamines, alkalemia.
- Prolonged administration of K-free parenteral fluids.
- Total parenteral hyperalimentation with inadequate K replacement.
- Loss in excessive lower GI secretions such as diarrhea, colonic fistulas, VIPoma.
- Diuretics.

signs and symptoms

- Electrocardiogram (ECG) (see Figure 6-3): Flattened T waves, ST depression, U wave.
- Arrhythmias, signs of low voltage.

treatment

- Check Mg level first as hypomagnesemia is commonly associated with hypokalemia and must be corrected before/along with hypokalemia.
- Amount of K to be replaced can be conservatively estimated as: (4.0 – current K) × 100, in mEq.
  - Example: if current K is 3.1, give 90 mEq (total, not all at once!!!)
- In asymptomatic patient with K > 3.0 mEq/L, oral K replacement may be sufficient.
- No more than 40 mEq should be added to a liter of IV fluid since rapid K administration can cause fatal arrhythmias.
- Rate should not exceed 40 mEq/hr.
- May cause a burning sensation if given in peripheral IV. Using low flow rate of 10 mEq/hr or adding a small amount of lidocaine to the solution can decrease discomfort.

HYPERKALEMIA

definition

> 5 mEq/L.

causes

- Commonly due to renal failure.
- Rarely found when renal function is normal and usually causes a transient hyperkalemia, due to cellular shifts: Potassium spillage from cells in severe injury; cells take up hydrogen ions in exchange for intracellular potassium, acting as a buffer in states of acidosis.
- Drugs: Angiotensin-converting enzyme (ACE) inhibitors, potassium-sparing diuretics.
- Iatrogenic causes: Penicillin G contains 1.7 mEq K per 1 million units, KCl added to maintenance fluids, blood transfusion with old batch of packed red blood cells (PRBCs) where K may have leaked out of cells, overtreatment of hypokalemia.
- Digoxin toxicity can cause severe hyperkalemia by blocking the sodium–potassium–adenosine triphosphatase (ATPase) pump.
- Hypoaldosteronism.
- Pseudohyperkalemia: Can result when RBCs lyse in the test tube and release potassium. This is a lab error: Repeat test before treating!

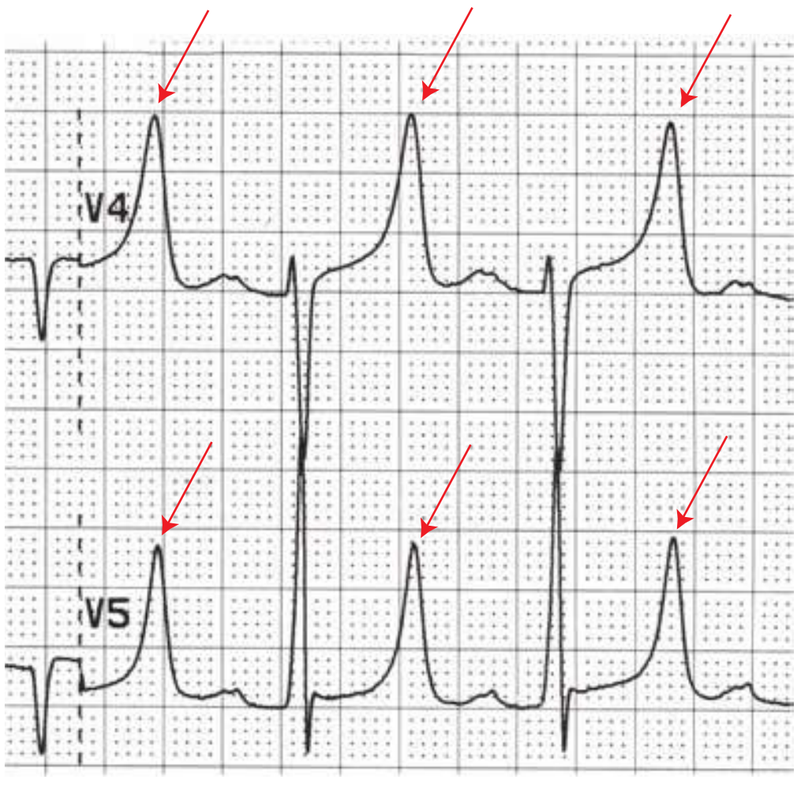


FIGURE 6-4. **Peaked t waves in hyperkalemia.**

signs and symptoms

Cardiac effects are most significant. Confirm hyperkalemia and obtain an ECG.

- ECG:
  - Early: Peaked T waves (see Figure 6-4), wide QRS, ST depression.
  - Late: Disappearance of T waves, heart block, sine wave—ominous for impending fatal arrhythmia, cardiac arrest.
- GI: Nausea, vomiting, intermittent intestinal colic, diarrhea.

treatment (in order of importance)

- Ten percent calcium gluconate 1 g IV—monitor ECG. Calcium temporarily suppresses cardiac arrhythmias by stabilizing the cardiac membrane and should be administered first. Does not effect potassium load.
- Lower extracellular K^+ (acute treatment): Albuterol, insulin with glucose or sodium bicarbonate promote cellular reuptake of K—transient relief of hyperkalemia.
- Kayexalate—cation exchange resin. As opposed to above measures, which immediately protect against dangers of high potassium, this actually removes the potassium from the body.
- Dialysis (last resort).

WARD TIP

How does Ca^+ help with hyperkalemia? It stabilizes the membrane potential of cardiac muscle, which would abnormally fire in the presence of high potassium. It does not actually have any effect on the amount of K^+ present.

Calcium Balance

- Normal: 1,000–1,200 mg—most is in the bone in the form of phosphate and carbonate.
- Normal daily intake: 1–3 g.
- Most excreted via stool (–200 mg via urine).
- Normal serum level: 8.5–10.5 mg/dL (total calcium).
- Half of this is nonionized and bound to plasma protein.

If hypocalcemia is seen on laboratory report, first correct for low albumin:

$$\text{Corrected Calcium} = 0.8\,(\text{Normal Albumin} - \text{Observed Albumin}) + \text{Observed Calcium}$$

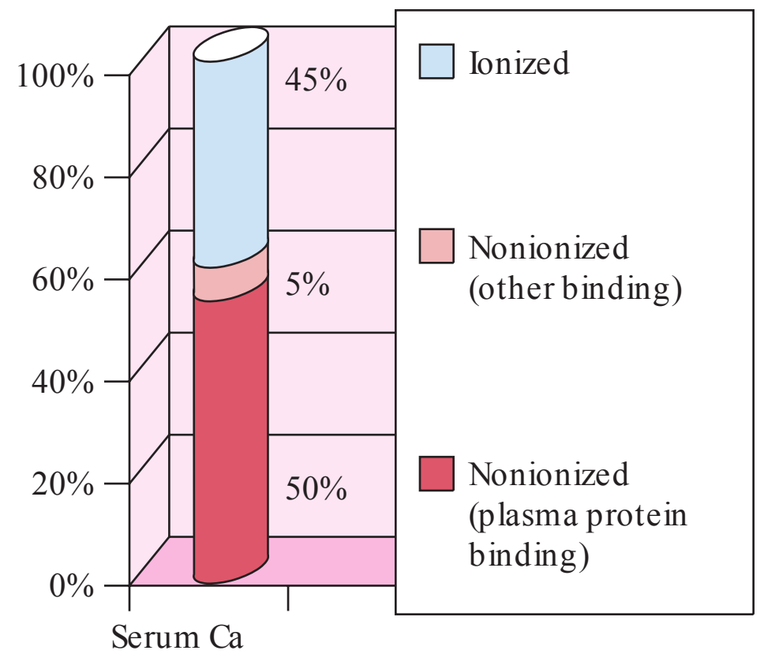


FIGURE 6-5. concentration of ionized and nonionized calcium in serum.

If corrected calcium falls within normal range, no action is required.

- Ionized calcium is the most accurate measure of calcium, but labs report total calcium.
- An additional nonionized fraction (5%) is bound to other substances in the ECF.
- Ratio of ionized to nonionized Ca is related to pH (see Figure 6-5):
  - Acidosis causes increase in ionized fraction.
  - Alkalosis causes decrease in ionized fraction.

hypocalcemia

definition

< 8 mg/dL.

causes

- Acute pancreatitis.
- Massive soft-tissue infections (necrotizing fasciitis).
- Acute/chronic renal failure.
- Pancreatic/small bowel fistulas.
- Hypoparathyroidism (common after parathyroid or thyroid surgery).
- Hypoproteinemia (often asymptomatic, corrected calcium will fall within normal range).
- Severe depletion of magnesium.
- Severe alkalosis may elicit symptoms in patient with normal serum levels because there is a decrease in the ionized fraction of total serum calcium.
- Recent massive pRBC transfusion (citrate in packed RBCs binds serum calcium)

signs and symptoms

- Numbness and tingling of fingers, toes, and around mouth.
- Increased reflexes.
- Chvostek's sign: Tapping over the facial nerve in front of the tragus of the ear causes ipsilateral twitching.
- Trousseau's sign: Carpopedal spasm following inflation of sphygmomanometer cuff to above systolic blood pressure for several minutes.
- Muscle and abdominal cramps.
- Convulsions.
- ECG—prolonged QT interval.

treatment

- IV Ca gluconate or Ca chloride.
- Monitor QT interval on ECG.

hypercalcemia

definition

> 15 mg/dL.

causes

- Hyperparathyroidism.
- Cancer (especially breast, multiple myeloma).
- Drugs (e.g., thiazides).

signs and symptoms

- Fatigue, weakness, anorexia, weight loss, nausea, vomiting.
- Somnambulism, stupor, coma.
- Severe headache, pain in the back and extremities, thirst, polydipsia, polyuria.
- Death.

treatment

- Vigorous volume repletion—dilutes Ca and increases urinary Ca excretion:
  - May be augmented with furosemide.
  - Definitive treatment of acute hypercalcemic crisis in patients with hyperparathyroidism is immediate surgery.
- Treat underlying cause.

WARD TIP

- Indications for emergency dialysis
  - **a :** Acidosis (refractory)
  - **E:** Electrolyte abnormalities (e.g., hyperkalemia)
  - **i:** Intoxication (to remove medications/drugs)
  - **o :** Overload (fluids)
  - **u :** Uremia (refractory or symptomatic, e.g., pericarditis)

Indications for Nutritional Support

enteral

- Gut works but oral intake not possible—altered mental state, ventilator, oral/pharyngeal/esophageal disorders.
- Oral intake not sufficient for metabolic requirements.
- Presence of malnutrition and wasting.

total parenteral nutrition (tpn)

- Enteral feeding not possible—GI obstruction, ileus.
- Enteral intake not sufficient for metabolic requirements—chronic diarrhea/emesis, malabsorption, fistulas, chemotherapy, irradiation therapy.
- The biggest danger of TPN is infection (the organic products in TPN can become infiltrated with bacteria and sent directly into the bloodstream).
- Adjunctive support necessary for managing disease—pancreatitis, hepatic failure, renal failure, chylothorax.
- Increased risks of acalculous cholecystitis, liver dysfunction.

Nutrient Requirements

caloric requirements

- Harris–Benedict equation and Fick equation (in patients with Swan–Ganz catheters) used to estimate basal energy expenditure (BEE).
- General estimation of BEE:
  - Males: BEE = **25 kcal/kg/day**
  - Females: BEE = **22 kcal/kg/day**
  - Multiply this by the desired goal:
    - Nonstressed patient: BEE × 1.2
    - Postsurgery: BEE × 1.3 to 1.5
    - Trauma/sepsis/burns: BEE × 1.6 to 2.0
    - Fever: 12% increase per °C
- The caloric needs must be met by nonprotein calories (i.e., fat and carbohydrate). Usually, carbs provide 70% of calories and fat provides 30%.

respiratory quotient (rq) of individual substrates

RQ is defined as the ratio of carbon dioxide released to oxygen consumed per unit metabolism of a substrate (i.e., VCO_2/VO_2).

- RQ for lipid: 0.7
- RQ for protein: 0.8
- RQ for carbohydrates: 1.0
- RQ for balanced diet: 0.83
- RQ for overfeeding: > 1

protein requirement

- Usual protein requirement is 0.8–1 g/kg body weight.
- Requirement increases in illness and is maximal in burn patients.
- The average surgical patient needs 1.2–1.6 g/kg protein intake.
- Each gram of urinary nitrogen is equivalent to 6.25 g of degraded protein.
  - Nitrogen balance:

    N Balance (g) = (Protein Intake (g)/6.25) – (UUN + 4)

    - UUN = Urinary urea nitrogen, 4 represents daily nitrogen loss other than UUN.
    - The goal of nitrogen balance is to maintain positive balance of 4–6 g.
- Vitamins and trace elements must be incorporated in all feeding regimens.

EXAM TIP

Causes of elevated anion gap metabolic acidosis:

MuDPilEs
Methanol/**M**etabolism (inborn errors)
uremia
diabetic ketoacidosis
Paraldehyde
iron/**i**soniazid
lactic acidosis
Ethylene glycol
salicylates/**s**trychnine

Narrow down to four basic processes:
Ketoacidosis
Lactic acidosis
Renal failure
Intoxication

Enteral Nutrition

- Preferred over parenteral nutrition.
- Reduces incidence of infection.
- May be continuous or intermittent.
- Routes for GI feeding: PO; NG feeding, gastrostomy, jejunostomy.
- Glutamine is the fuel for enterocytes.
- Short-chain fatty acids serve as fuel for the colonocytes.
- Omega-3 fatty acids and arginine serve as immune-modulating agents.

Complications

- Diarrhea.
- Aspiration.
- Obstruction of feeding tube.

Acid–Base Disorders

Assess the acid–base disorder step by step (Table 6-5):

- Is the primary disorder an acidosis (pH < 7.40) or alkalosis (pH > 7.40)?
- Is the disorder respiratory (pH and Pco_2 move in opposite directions)?
- Is the disorder metabolic (pH and Pco_2 move in same direction)?
- Is the disorder a simple or mixed disorder?

Use the following general rules of thumb for acute disorders:

- Metabolic acidosis: Pco_2 drops ~ 1.5 (drop in HCO_3).
- Metabolic alkalosis: Pco_2 rises ~ 1.0 (rise in HCO_3).
- Respiratory acidosis: HCO_3 rises ~ 0.1 (rise in Pco_2).
- Respiratory alkalosis: HCO_3 drops ~ 0.3 (drop in Pco_2).

Compensation beyond above parameters suggests mixed disorder.

Metabolic Acidosis

- Two varieties:
  - **Anion gap acidosis**—due to addition of unmeasured acid.
  - **Nonanion gap acidosis**—due to HCO_3 loss.

TABLE 6-5. Respiratory and Metabolic Components of Acid–Base Disorders

	Acute (uncompensated)			Chronic (partially compensated)		
Type of Acid–Base Disorder	pH	Pco_2 (respiratory component)	Plasma HCO_3^{-a} (metabolic component)	pH	Pco_2 (respiratory component)	Plasma HCO_3^{-a} (metabolic component)
Respiratory acidosis	↓↓	↑↑	N	↓	↑↑	↑
Respiratory alkalosis	↑↑	↓↓	N	↑	↓↓	↓
Metabolic acidosis	↓↓	N	↓↓	↓	↓	↓
Metabolic alkalosis	↑↑	N	↑↑	↑	↑?	↑

[a]Measured as standard bicarbonate, whole blood buffer base, CO_2 content, or CO_2 combining power. The base excess value is positive when the standard bicarbonate is above normal and negative when the standard bicarbonate is below normal.

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 51.

EXAM TIP

Causes of normal anion gap metabolic acidosis:

HARD UP
Hyperparathyroidism
Adrenal insufficiency/**A**nhydrase (carbonic anhydrase) inhibitors
Renal tubular acidosis
Diarrhea
Ureteroenteric fstula
Pancreatic fstulas

- Calculating the anion gap:

$$AG = Na - [Cl + HCO_3]$$

  - Normal AG = 10

Metabolic alkalosis

Two mechanisms:

- Loss of H^+ from kidneys or GI tract:
  - Renal: Mineralocorticoid excess, diuretics, potassium-losing nephropathy.
  - GI: Vomiting, gastric drainage, villous adenoma of colon.
- Gain HCO_3: Milk-alkali syndrome, exogenous $NaHCO_3$; lactated Ringer's, packed RBCs, TPN all contain substrates that metabolize to bicarbonate.

respiratory acidosis

Hypercapnia secondary to one of two mechanisms:

- Hypoventilation (brain stem injury, neuromuscular disease, ventilator malfunction, opiates).
- Ventilation-perfusion (V/Q) mismatch (chronic obstructive pulmonary disease, pneumonia, pulmonary embolism, foreign body, pulmonary edema).

respiratory alkalosis

- Hyperventilation secondary to anemia, anxiety, increased intracranial pressure (ICP), salicylates, fever, hypoxemia, systemic disease (sepsis), pain, pregnancy, CHF, pneumonia, asthma, liver disease.
- Alkalosis causes decrease in serum K and ionized Ca, resulting in paresthesias, carpopedal spasm, and tetany.

WARD TIP

Causes of respiratory alkalosis:

Mis [Ha P]^{3}s
Mechanical overventilation
increased ICP
s epsis
Hypoxemia/**H**yperpyrexia/**H**eart failure
a nxiety/**a** sthma/**a** scites
Pregnancy/**P**ain/**P**neumonia
s alicylates

HIGH-YIELD FACTS IN

The esophagus

Anatomy and Physiology 92

Tools for the Assessment of Esophageal Functions 93

Esophageal Motor Disorders 93

- Definition 93
- Achalasia 93
- Diffuse esophageal spasm (DES) 94
- esophageal diverticula 95
- Zenker's Diverticulum 96

Esophageal Varices 96

- pathophysiology 96
- Signs and Symptoms 96
- treatment 97

Esophageal Perforation or Rupture 97

- Definition 97
- etiology 97
- Signs and Symptoms 98
- Diagnosis 98
- treatment 98

Caustic Injury 98

- Signs and Symptoms 99
- treatment 99

Gastroesophageal Reflux Disease (GERD) 99

- Definition 99
- pathophysiology 99
- Signs and Symptoms 100
- Diagnosis 100
- treatment 100

Barrett's Esophagus 102

- Definition 102
- Signs and Symptoms 102
- Diagnosis 102
- management 102

Esophageal Carcinoma 102

- epidemiology 102
- risk factors 103
- Signs and Symptoms 103
- Diagnosis 103
- treatment 104

Miscellaneous Esophageal Disorders 105

Anatomy and Physiology

The esophagus is a 25-cm-long muscular tube that begins at the pharynx (begins at lower border of C6), travels through the thorax in the posterior mediastinum, and empties into the cardia of the stomach.

- Superior third: Striated muscle only.
- Middle third: Both striated and smooth muscle.
- Inferior third: Smooth muscle only.

Three areas of narrowing (evident on barium swallow; see Figure 7-1):

1. At the beginning of the esophagus, caused by the cricopharyngeus muscle.
2. Where the left mainstem bronchus and aortic arch cross.
3. At the hiatus of the diaphragm.

Two sphincters are present, which function as control points:

- Upper esophageal sphincter (UES, cricopharyngeus muscle) prevents the passage of excess air into the stomach during breathing.
- Lower esophageal sphincter (LES) is a physiological sphincter; it relaxes with initiation of the pharyngeal swallow and prevents the reflux of gastric contents when swallowing is not occuring.
- Esophageal peristalsis accompanying swallowing is termed primary peristalsis.
- Secondary peristalsis can be initiated by the esophageal musculature without the pharyngeal phase to clear the esophagus of any substance left behind from primary peristalsis.

WARD TIP

The esophagus does not have the serosal layer, so esophageal anastomoses are prone to leaks.

WARD TIP

The distance of the gastroesopohageal (GE) junction from the incisor teeth is 40 cm and serves as an important landmark in upper gastrointestinal (GI) endoscopy.

WARD TIP

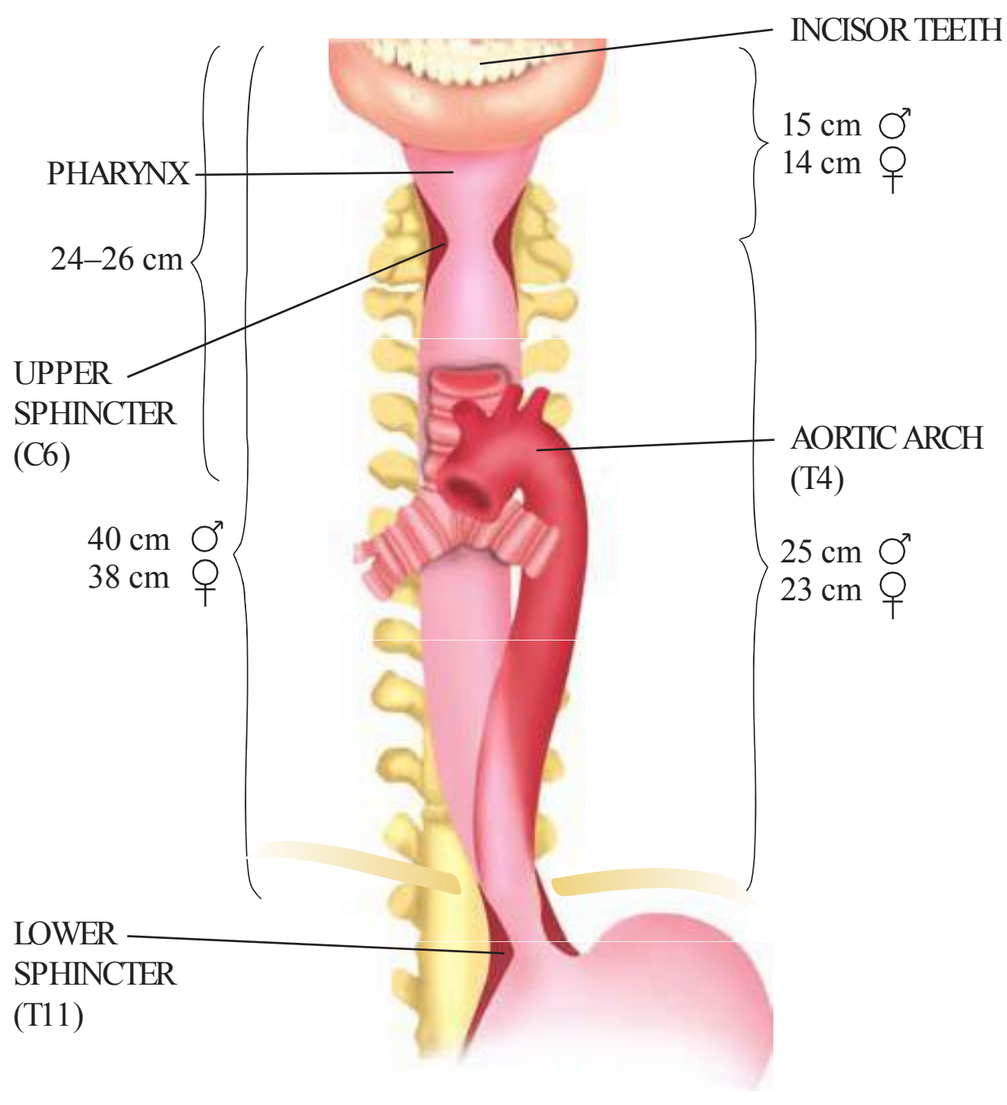
Clinically significant motility disorders involve smooth muscle in the lower two thirds only.

EXAM TIP

Recall the vertebral levels at which the following traverse the diaphragm:

T8 = inferior vena cava (IVC).
T10 = esophagus.
T12 = aorta.

"I (IVC) ate (T8) ten (T10) eggs (esophagus) at (aorta) noon (T12)."



FIgu Re 7-1. Important clinical endoscopic measurements of the esophagus in adults.

Tools for the Assessment of Esophageal Functions

- Barium swallow: The patient ingests a radiopaque substance; swallowing and the esophagus are analyzed fluoroscopically.
- Flexible endoscopy: A camera is passed through the oral cavity into the esophagus, with the patient under sedation. There are multiple ports in the scope to allow for biopsies, irrigation, and suction.
- Manometry: Examines the motor function.
- Twenty-four-hour ambulatory pH monitoring.

Esophageal Motor Disorders

Result from abnormalities in the propulsive pump action of the esophagus or relaxation of the LES. Can be primary (achalasia, diffuse esophageal spasm [DES], nutcracker esophagus, hypertensive LES) or secondary (result of another systemic disease, including collagen vascular diseases like scleroderma and mixed connective tissue disease, neuromuscular disease, endocrine and metastatic disorders).

Definition

- Dysphagia: Difficulty swallowing.
- Odynophagia: Pain on swallowing. May or may not accompany dysphagia.

Achalasia

Definition

Failure of the lower portion of the esophagus to relax during swallowing is defined as achalasia. The resulting dysphagia is due to three mechanisms:

1. Complete absence of peristalsis in the esophageal body.
2. Incomplete/impaired relaxation of the LES after swallowing.
3. Increased resting tone of the LES.

These result in elevation of intraluminal esophageal pressure, esophageal dilatation, and subsequent progressive loss of normal swallowing mechanisms—a functional holdup of ingested material.

Signs and Symptoms

- Dysphagia for both solids and liquids.
- Regurgitation of food.
- Severe halitosis (due to the decomposition of stagnant food within the esophagus).

Diagnosis

- Lateral upright chest x-ray (CXR) may reveal a dilated esophagus and the presence of air-fluid levels in the posterior mediastinum.
- Barium swallow will reveal the characteristic distal bird's beak sign due to the collection of contrast material in the proximal dilated segment and the passage of a small amount of contrast through the narrowed LES (Figure 7-2).
- Esophageal motility study will confirm nonperistaltic contractions, incomplete LES relaxation, and increased LES tone.
- Esophagoscopy is indicated to rule out mass lesions or strictures, and to obtain specimens for biopsy.

WARD TIP

- A complaint of dysphagia must elicit a full dietary history from a patient; what they experience, when they eat, what types of food they eat that cause it.
- If the patient is immunocompromised, have a high index of suspicion for candidal esophagitis if human immunodeficiency virus (HIV)-positive, and for cytomegalovirus (CMV)/herpes simplex virus (HSV) esophagitis if non-HIV.

WARD TIP

achalasia: Gr. Failure to relax.

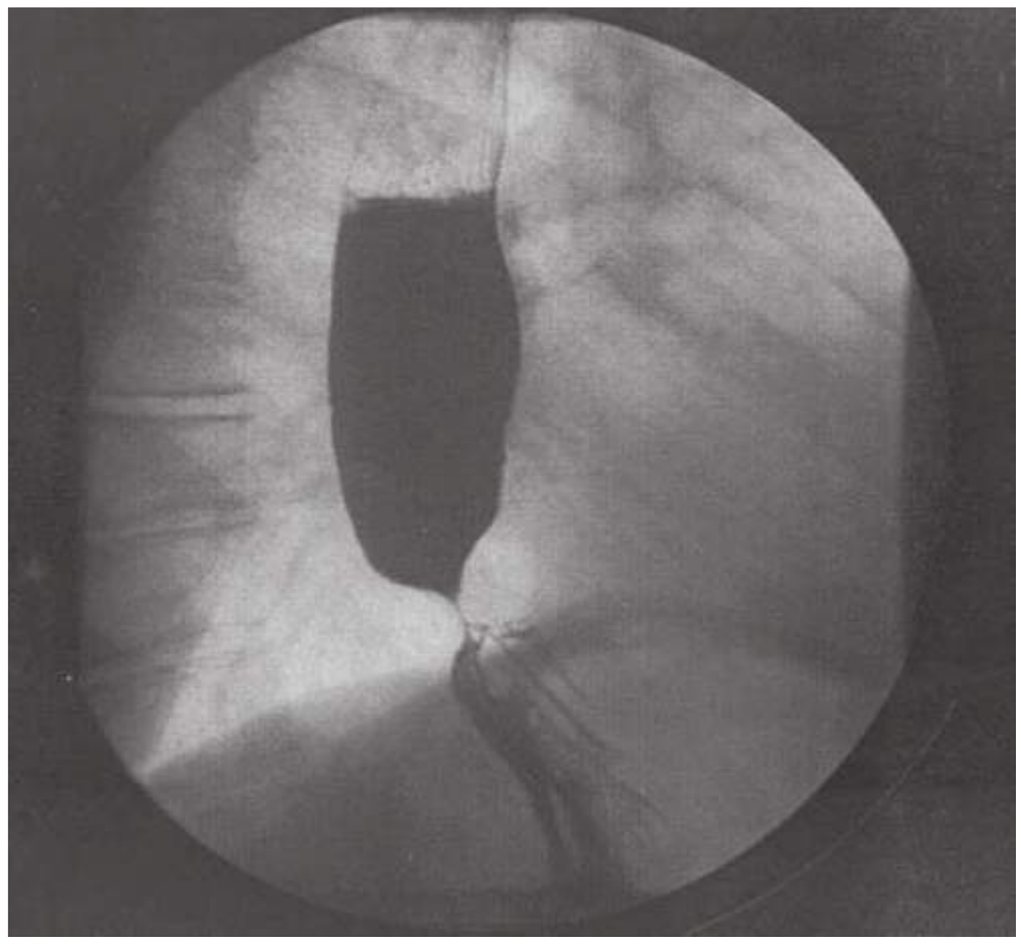
EXAM TIP

Classic triad of achalasia: Dysphagia to solids and liquids, regurgitation, and weight loss.

EXAM TIP

Barium swallow:

- Bird's beak or steeple sign: Achalasia.
- Corkscrew-shaped: Diffuse esophageal spasm.



FIgu Re 7-2. Barium study demonstrating a dilated esophagus and smooth, tapered gastroesophageal junction, typical of achalasia. (Reproduced, with permission, from Woltman T, Oelschlager BK. Benign esophageal disorders. In Zinner MJ, Ashley SW, eds. Maingot's Abdominal Operations. 11th ed. New York: McGraw-Hill; 2007: 212.)

EXAM TIP

Gastroesophageal reflux produces a sour taste and a gush of saliva due to the presence of hydrochloride from the gastric acid (water brash), while achalasia does not.

WARD TIP

Esophageal perforation is four times more likely following dilatation compared to esophagomyotomy.

Treatment

- Medical management: Drugs that relax the LES—nitrates, calcium channel blockers, and antispasmodics.
  - Botox injection: Helps relax the LES.
  - Endoscopic dilatation: Has a lower success rate and a higher complication rate. It involves inserting a balloon or progressively larger sized dilators through the narrowed lumen, which causes tearing of the esophageal smooth muscle and decreases the competency of the LES.
- Surgical management: Esophagomyotomy (Heller's myotomy) with or without fundoplication is the treatment of choice for achalasia.
- Esophagomyotomy: Esophagus is exposed via a transthoracic (left thoracotomy), transabdominal, thorascopic, or laparoscopic technique. The tunica muscularis of the esophagus is incised distally, with extension to the LES. Complete division of the LES necessitates the addition of an antireflux procedure a (partial fundoplication).

Complications

- Risk of squamous cell carcinoma (SCC) is as high as 10% in patients with long-standing achalasia (duration 15–25 years).
- Patients may also develop pulmonary complications such as aspiration pneumonia, bronchiectasis, and asthma due to reflux and aspiration.

Diffuse esophAgeAl spAsm (Des)

Definition

DES is a disorder of unknown etiology that is primarily a disease of the esophageal body. It may be a primary disease process of the muscle, or may occur in association with reflux esophagitis, esophageal obstruction, collagen vascular disease, or diabetic neuropathy. Spasm occurs in the distal two thirds of the esophagus and is caused by uncoordinated large-amplitude rapid contractions of smooth muscle. By def nition, the LES tone is normal.

signs and symptoms

- Dysphagia for both solids and liquids.
- Substernal chest pain, similar to that seen in a myocardial infarction: Acute onset of severe retrosternal pain that may radiate to the arms, jaw, or back. The chest pain may occur at rest, or it may follow swallowing. The degree of chest pain depends on the duration and severity of the contractions.
- No regurgitation (unlike achalasia); no water brash (unlike gastroesophageal reflux disease).

Diagnosis

- Barium swallow may reveal the characteristic "corkscrew" appearance of the esophagus, due to the ripples and sacculations that are visible as a result of uncoordinated esophageal contraction. Barium swallow may be entirely normal, however, because the esophagus may not be in spasm at the time of the study. In contrast to achalasia, the LES appears to be a normal diameter.
- Esophageal manometry studies will reveal the presence of large, uncoordinated, and repetitive contractions in the lower esophagus. Alternatively, manometry may appear normal when the patient is asymptomatic. LES manometry will show normal resting pressure with LES relaxation upon swallowing (again, unlike achalasia).
- Esophagoscopy should be performed to rule out mass lesions, strictures, or esophagitis.

Treatment

- Nitrates or calcium channel blockers to relax smooth muscle.
- Surgical treatment via an esophageal myotomy is not as successful in relieving symptoms as it is for achalasia, and is therefore not recommended unless dysphagia is severe and incapacitating (see Table 7-1).

esophAgeAl DiverticulA

Definition

- Outpouching of the esophageal mucosa that protrudes through a defect in the muscle layer. Often occur when there are coexistent motility disorders.

TaBLe 7-1. achalasia vs. Diffuse esophageal spasm

	AchAlAsiA	DiffuseesophAgeAl SpAsm
signs and symptoms	Dysphagia, regurgitation of undigested food, severe halitosis, weight loss, cough, diffuse chest pain.	Dysphagia, diffuse chest pain.
pattern of contraction	Failure of LES to relax on swallowing.	Swallow-induced large wave of esophageal contraction, normal LES pressure.
Barium swallow findings	Absence of gastric bubble, narrowing of terminal esophagus that looks like a bird's beak.	Corkscrew appearance.
Treatment	Nitroglycerin, local botulinum toxin, balloon dilatation, sphincter myotomy (Heller's myotomy)	Nitroglycerin, nifedipine; surgery only if symptoms are severe and persistent.

WARD TIP

Due to the fact that DES produces cardiac-like complaints, the diagnosis is often delayed until an extensive cardiologic workup is performed.

WARD TIP

Patients with DES often have other functional intestinal disorders such as irritable bowel syndrome and spastic colon.

WARD TIP

Nutcracker esophagus, another hypermotility disorder, involves more focal segments of the esophagus.

EXAM TIP

epi**p**hrenic and **p**haryngoesophageal = caused by **e**levated **p**ressure (**p**ulsion) and are **p**seudo (false).

- May be either a true diverticulum, which involves all three layers of the esophagus (e.g., midesophageal diverticulum), or a false diverticulum, involving only the mucosa and submucosa (e.g., Zenker's diverticulum).
- Characterized by its location: Pharyngoesophageal (Zenker's diverticulum), midesophageal, or epiphrenic (terminal third of the esophagus).
- Pharyngoesophageal/Zenker's and epiphrenic diverticula are called pulsion diverticula, since they are caused by increased esophageal pressure; they are false diverticula.

Zenker's Diverticulum

Signs and Symptoms

Pharyngoesophageal (Zenker's) type is the most likely to be symptomatic. Typical symptoms include dysphagia along with spontaneous regurgitation of undigested food, halitosis, choking, aspiration, repetitive respiratory infections, and, eventually, debilitation and weight loss.

WARD TIP

When **Z**enker's causes **Z**ymptoms it requires **Z**urgery. Asymptomatic Zenker's is treated only if it is > 2 cm size.

Diagnosis

A barium swallow will reveal the presence of all types of diverticula. Endoscopy is difficult and potentially dangerous due to the risk of perforation through the diverticulum.

Treatment

Treatment of Zenker's diverticulum is recommended to relieve symptoms and to prevent complications.

- The most common procedure is a cervical pharyngocricoesophageal myotomy (incising the cricopharyngeus) and is done in all cases needing operative intervention.
- Diverticulopexy (suturing the diverticulum in the inverted position to the prevertebral fascia) is added to myotomy for larger diverticula.
- Diverticulectomy (endoscopic stapling of the diverticulum), along with myotomy, is performed for the largest diverticulae.

WARD TIP

- When operating on a Zenker's diverticulum, the most important part of the procedure to prevent recurrence is the myotomy, **No T** the diverticulectomy.

Esophageal Varices

Pathophysiology

- Occur as a result of portal hypertension, most commonly a result of alcoholic cirrhosis.
- As elevated portal system pressure impedes the flow of blood through the liver (increased intrahepatic resistance), various sites of venous anastomosis become dilated secondary to retrograde flow from the portal to systemic circulations. Varices are portosystemic collaterals.
- Clinically significant portal-systemic sites:
  - Cardio-esophageal junction—dilatation leads to esophageal varices.
  - Periumbilical region—dilatation leads to caput medusae.
  - Rectum—dilatation leads to hemorrhoids.
    - Accounts for 10–30% of upper GI bleeds; up to 30% are fatal, 70% rebleed.

Signs And Symptoms

- Painless hematemesis.
- Unprovoked (i.e., not postemetic).

- Hemodynamic instability is common.
  - Risk for rebleeding is high.
  - Peripheral stigmata of liver disease.

treatment

- Identifying high-risk patients and preventing the first bleeding episode are critical (i.e., screening endoscopy to determine varices in cirrhotic patients). This includes pharmacological therapy to reduce portal pressure and consequently intravariceal pressure—reduce collateral portal venous flow with vasoconstrictors (somatostatin, vasopressin and octreotide decrease portal flow) and intrahepatic resistance with vasodilators (beta blockers, especially propranolol and nitrates, decrease portal pressure).
  - Variceal bleeding ceases spontaneously in ~50% of cases.
- Management of ruptured varices causing an acute bleed:
  1. Stabilization of hemodynamics: Volume replacement with normal saline (NS) or lactated Ringer's and packed red blood cells (PRBCs), nasogastric (NG) suction, and lavage.
  2. Continuous vasopressin/somatostatin/octreotide to reduce splanchnic blood flow and portal pressure.
  3. Urgent endoscopic therapy: Endoscopic sclerotherapy (injection of the bleeding vessel(s) with a sclerosing agent via a catheter that is passed through the endoscope) stops bleeding in 80–90%; endoscopic band ligation (small elastic band is placed around the bleeding varix resulting in hemostasis) is equivalent to sclerotherapy initially, with fewer complications.
  4. Balloon tamponade (Sengstaken–Blakemore tube) to apply direct pressure and hemostasis to the varix with an inflatable balloon.
  5. For refractory acute bleeding, TIPSS procedure (transjugular intrahepatic portosystemic shunt).
  6. Intraoperative placement of a portocaval shunt. Surgical therapy is considered when there is continued hemorrhage or recurrent rebleeding with poor control.
  7. Liver transplant.

WARD TIP

Endoscopic sclerotherapy or band ligation for control of ruptured esophageal varices has a 90% success rate. Patients are usually intubated prior to the procedure to prevent aspiration of blood.

EXAM TIP

- Medical management in esophageal varices: Beta-blockers (propranolol, nadolol) and nitrates in asymptomatic patients; octreotide in active bleed.

EXAM TIP

The most serious complication of balloon tamponade for esophageal varices is esophageal perforation.

EXAM TIP

- TIPS can worsen hepatic encephalopathy—treat with lactulose, neomycin, or rifaximin.

Esophageal Perforation or Rupture

Definition

- Trauma to the esophagus that may result in leakage of air and esophageal contents into the mediastinum.
- A surgical emergency.
- Carries a 50% mortality.

etiology

- The most common cause of esophageal perforation is iatrogenic. Occurs following endoscopy, dilatation, tamponade tubes (Blakemore, Minnesota).
- Boerhaave syndrome (15% of cases): A spontaneous perforation and full-thickness tear. Usually occurs in the area of the left pleural cavity or just above the GE junction, due to transmission of abdominal pressure to the esophagus. Can result from forceful vomiting, retching coughing, labor, lifting, or trauma.

EXAM TIP

Typical scenario: A man presents severe retrosternal and upper abdominal pain after an episode of retching. Think: Boerhaave syndrome (full-thickness) or Mallory–Weiss syndrome (partial-thickness) tears in the esophagus.

A patient who recently underwent an endoscopic procedure develops fever and chest pain. Think: Iatrogenic esophageal rupture.

- Mallory–Weiss syndrome: A partial-thickness mucosal tear. Usually occurs in the right posterolateral wall of the distal esophagus and results in bleeding that generally resolves spontaneously. Due to forceful vomiting.
- Foreign body ingestions (14% of cases): Objects usually lodge near anatomic narrowings and then perforate through:
  - Above the UES.
  - Near the aortic arch.
  - Above the LES.

WARD TIP

In Boerhaave syndrome, the most common site of rupture is the left lateral wall of the esophagus, just above the esophageal hiatus. Iatrogenic perforation occurs most commonly following esophagogastroduodenoscopy (EGD) in the cervical esophagus near the cricopharyngeus muscle.

Signs and Symptoms

- Severe, constant cervical, substernal, or back pain (depending on the location of the perforation).
- Dysphagia.
- Dyspnea.
- Subcutaneous emphysema.
- Mediastinal emphysema heard as a "crunching" sound (Hamman's sign).
  - Sepsis/fever.
  - Pneumothorax.

Diagnosis

- CXR: Left-sided pleural effusion; mediastinal, cervical, or subcutaneous emphysema; mediastinal widening.
- Esophagogram with water-soluble contrast (gastrografin): Shows extravasation of contrast in 90% of patients.
- If water-soluble (gastrografin) study is negative, the patient must then get a barium swallow before a perforation can be ruled out.
- Other studies: Endoscopy, computed tomography (CT), and thoracentesis (check fluid for low pH and high amylase).

WARD TIP

Subcutaneous and mediastinal emphysema signify a full-thickness tear.

Treatment

Resuscitation and stabilization of patient.

- Primary surgical closure of full-thickness tears within 24 hours: 80–90% survival rate.
- Drain the contaminated mediastinum.
- Monitor for recovery from sepsis.
- Conservative nonoperative management: If the perforation is well contained in the mediastinum (with the barium draining back into the esophagus) and the patient has mild symptoms with minimal signs of sepsis, then the perforation can be managed with hyperalimentation, antibiotics, and gastric acid inhibition. Oral intake can be resumed within 1–2 weeks.

Caustic Injury

- Caused by acid (in household cleaning agents) or alkali (lye, sodium hydroxide tablets).
- Alkali burns are worse than acidic, as acid substances usually burn the mouth immediately and are less frequently swallowed; alkaline substances are more frequently ingested. Acidic substances also cause coagulative tissue necrosis, which limits their penetration, whereas alkaline substances cause injury deep into the tissue as they dissolve the tissue.

- Caustic injury has both an acute phase—controlling immediate tissue injury and perforation potential—and a chronic phase—managing structures and swallowing disturbances that have developed.
- Acute damage is dependent on nature of substance, quantity, and time in contact with tissue.

Signs and Symptoms

- Oral and substernal pain in the initial phase; pain on swallowing and dysphagia.
- Hypersalivation.
- Fever—strongly correlated with an esophageal lesion.
- Vomiting and hemoptysis.
- Systemic hypovolemia and acidosis.
- Laryngospasm, laryngedema.
- Dysphagia reappears in the chronic phase due to fibrosis, retraction, and narrowing of the esophagus. Strictures develop in 80% of patients within 2 months.

Treatment

- Careful inspection of both the oral cavity and esophagus; however, there is poor correlation between the appearance of one and damage to the other.
- Early esophagoscopy (< 24 hours) to establish the presence of esophageal injury with exquisite care not to perforate the already damaged esophagus.
- Limiting the burn by administering neutralizing agents, preferably within the first hour. Lye/alkali can be neutralized with half-strength vinegar, lemon juice, or orange juice; acid with milk, egg white, or antacid.
- Broad-spectrum antibiotics to prevent infectious complications.
- Dilatations are controversial as they can traumatize the esophagus, but some start them early after injury to preserve the esophageal lumen and remove adhesions. They are done with a bougie in order to prevent and manage strictures.
- Extensive necrosis leading to perforation is best managed by resection; if the esophagus is viable, it can be managed with an intraluminal esophageal stent.
- Surgical intervention is indicated if there is complete stenosis with failure to establish a lumen, marked irregularity on barium swallow, development of severe mediastinitis with dilatation, fistula formation, or if the patient is unable to undergo prolonged periods of dilatation.
- Currently, the stomach, jejunum, and colon are organs used to replace the esophagus.

EXAM TIP

Emetics are **contraindicated**—forceful vomiting can cause perforation.

Gastroesophageal Reflux Disease (GERD)

Definition

- Defined by symptoms, presence of endoscopic esophagitis, or by measuring the increased exposure of the esophagus to gastric juice.
- Common disease, accounts for ~75% of esophageal pathology.

Pathophysiology

- Loss of the normal gastroesophageal barriers results in reflux. The primary barrier is the LES, and it is usually secondary to low or reduced LES resistance with reflux of acidic gastric contents into the esophagus.

- Causes include a structurally defective sphincter; hiatal hernia; transient loss of the GE barrier (with a structurally normal LES) secondary to gastric abnormalities such as distention with air or food; delayed gastric emptying; and increased intra-abdominal pressure.
- Prolonged exposure to a low pH from gastric contents (acid, pepsin, and duodenal contents, including biliary and pancreatic secretions) will cause irritation of the esophageal mucosa (as well as the respiratory epithelium) and the development of complications including esophagitis, stricture, Barrett's esophagus (see next section) and risk of aspiration.

signs And symptoms

- Patients with GERD may report a range of symptoms from heartburn to angina-like chest pain.
  - Atypical symptoms include nausea, vomiting, postprandial fullness, choking, chronic cough, wheezing, and hoarseness.
- Minimal or transient reflux may cause asymptomatic esophagitis, while severe reflux may cause severe esophagitis accompanied by laryngitis, aspiration pneumonitis/recurrent pneumonia, idiopathic pulmonary fibrosis, or asthma.
- The presence of dysphagia may indicate peptic stricture formation.

Diagnosis

- Patients presenting with vague symptoms of chest pain must be evaluated for cardiac and pulmonary disease as deemed necessary (thorough physical exam, electrocardiogram [ECG], cardiac enzymes, and admission as appropriate).
- A barium study is useful to look for an anatomical cause for reflux, such as a hiatal hernia; can also elucidate pathology resulting from long-standing reflux, such as a stricture or ulcer formation.
- Twenty-four-hour ambulatory pH monitoring of the esophagus: A probe with pH electrodes is inserted into the patient's esophagus for 24 hours. The probe continuously records the esophageal pH; useful in determining the severity of reflux (gold standard for diagnosing GERD).
- Esophageal manometry is useful in evaluating the competence of the LES.
- Esophagoscopy should be performed to evaluate the esophageal mucosa to rule out Barrett's esophagus, and to obtain specimens for biopsy and Helicobacter pylori testing.

WARD TIP

Twenty-four-hour pH monitoring of the esophagus is the gold standard for diagnosing GERD.

treatment

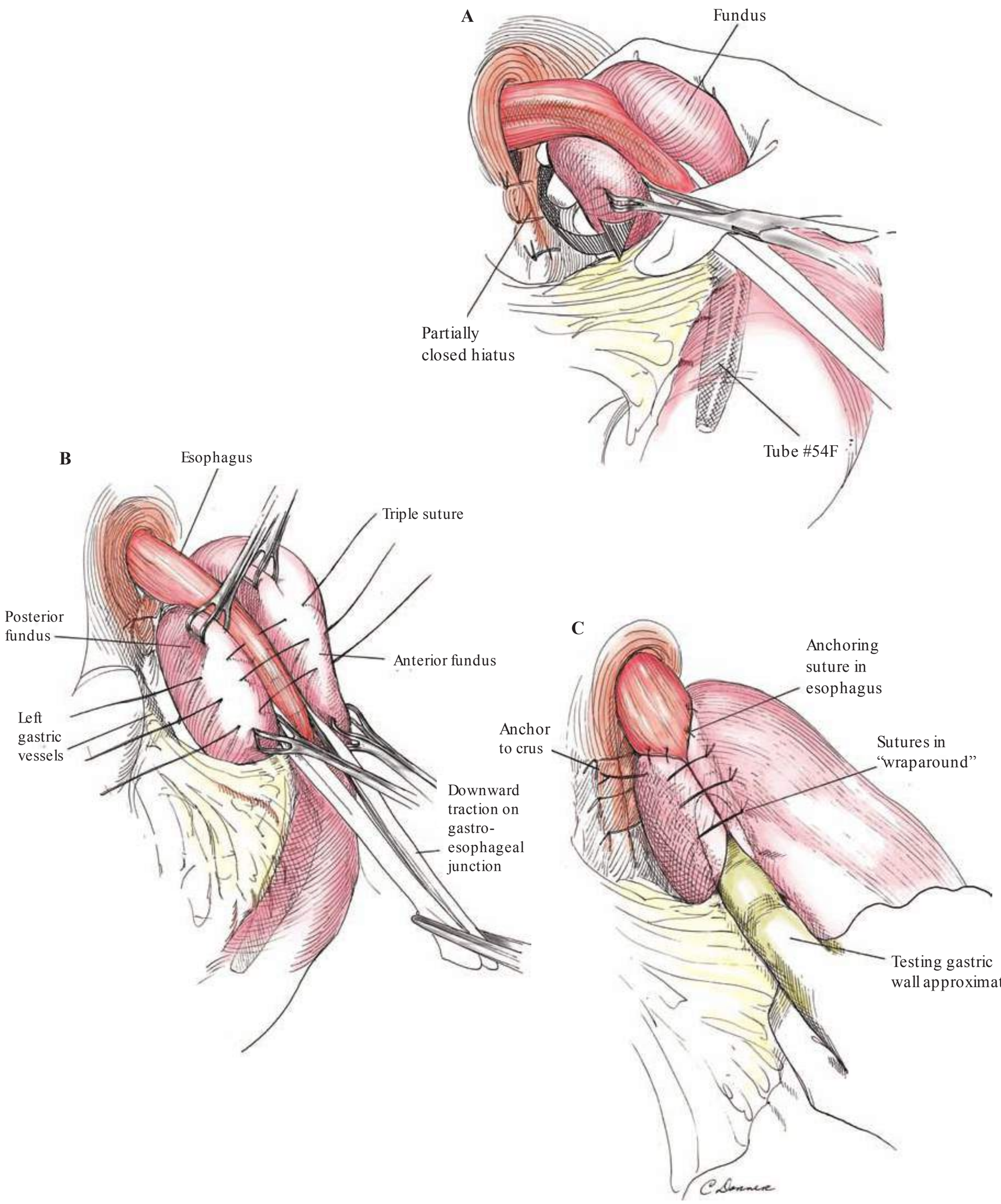
Initial treatment of GERD involves medications that decrease gastric acid production along with lifestyle modification, including:

- Elevation of head end of bed.
- Antacids (symptomatic relief).
- H_2 antagonists (e.g., ranitidine).
- Proton pump inhibitors (PPIs; e.g., omeprazole).
- Education to avoid alcohol, coffee, chocolate, and peppermint, as they may aggravate symptoms; avoid nicotine, as it decreases LES tone.
- Instruct and educate the patient to eat small, frequent meals, elevate the head of the bed, avoid tight clothing, and to not go to sleep within 3–4 hours of a meal.
- Medications that promote gastric emptying (e.g., metoclopromide) may be beneficial early in the disease.
- The patient undergoes a trial of medical therapy for 6–12 weeks before further investigations.
- If medical management fails and the patient develops complications like chronic esophagitis or stricture, surgical intervention should be considered.

EXAM TIP

If multiple modalities are available for evaluating and treating the patient, always begin with the least expensive one (i.e., digital rectal exam for suspected prostate cancer, medication trial for suspected reflux disease).

- Surgery should be limited to those patients who have persistent or progressive disease despite maximal medical therapy or with a structurally defective LES. The primary goal of surgery is to return normal sphincter length and function and return the physiologic swallowing functions of the esophagus. The procedure of choice is a fundoplication (Figure 7-3)—wrapping



FIgu Re 7-3. Fundoplication procedure. A. A large gastric tube or rubber esophageal dilator is inserted into the esophagus to prevent undue compression of the esophageal lumen. The right hand is introduced behind the fundus of the stomach to test the adequacy of the gastric mobilization. **B.** One or more long Babcock forceps are applied to the gastric wall on either side of the esophagus. The anterior and posterior gastric walls are approximated with interrupted silk sutures. **C.** After the traction drain and esophageal dilator are removed, the surgeon introduces the index f nger or thumb upward under the plicated gastric wall. (Reproduced, with permission, from Zollinger RM Jr, Zollinger RM Sr. Zollinger's Atlas of Surgical Operations. 8th ed. New York: McGraw-Hill; 2003: 97.)

the fundus of the stomach around the distal portion of the esophagus to create a sphincter, called the Nissen procedure, can be open or laparoscopic, transabdominal or transthoracic.
- Treatment of GERD is important in order to prevent the progression to Barrett's esophagus. In addition, chronic reflux predisposes the patient to pulmonary complications as mentioned.

Barrett's Esophagus

Definition

A condition in which the distal portion of the tubular esophagus becomes lined by columnar epithelium as opposed to the normal squamous epithelium—histological appearance of intestinal metaplasia (the appearance of goblet cells).

This new region is susceptible to ulceration, bleeding, stricture, and adenocarcinoma formation.

Signs And Symptoms

- Usually similar to patients with GERD.
- Bleeding, hematemesis.
- Signs of esophageal perforation (see previous discussion).

Diagnosis

- Endoscopy for evaluation. Suspect when there is difficulty visualizing the squamocolumnar junction in the lower esophagus or an appearance of a redder mucosa.
- Multiple biopsies should be taken for a definitive or histologic diagnosis.

Management

- The same as those patients with GERD—require long-term PPI therapy for symptom relief and management of esophageal mucosal injury.
- Monitor and prevent disease progression to malignancy (risk 1% per year).
- Antireflux surgery when there are associated complications (stricture, ulceration, metaplastic progression).
- Surgical resection for refractory cases with high-grade dysplasia.

Esophageal Carcinoma

Epidemiology

- The increasing prevalence of adenocarcinomas (due to Barrett's) as compared to what was mostly SCC is shifting the epidemiology of esophageal cancer. Adenocarcinomas have increased in prevalence to more than 50% of all esophageal cancer in the western world. Other tumors of the esophagus are less common (including leiomyomas, melanomas, carcinoids, lymphomas).

- Most cases occur in patients over the age of 50, but there is an increase in cases in younger patients with disease detected at an earlier stage.
- Males are affected more frequently than females.
- Over 50% of patients have unresectable or metastatic disease at the time of presentation.
- Five-year survival rate is poor, but has increased slightly to 15%.

Risk Factors

- Environmental:
  - Tobacco.
  - Alcohol.
  - Food additives (nitrates in smoked and pickled meats).
- Esophageal disorders:
  - GERD/Barrett's esophagus.
  - Achalasia.
  - Damage from caustic ingestion/strictures:
    - Chronic esophagitis.
    - Plummer–Vinson syndrome.
- History of radiation therapy to the mediastinum.

Signs And Symptoms

- Physical exam is usually entirely normal.
  - Patients may present with nonspecific GI complaints.
  - Gradual development of dysphagia (74% of patients) due to invasion of serosal layer, first for solids and later for both solids and liquids (mechanical dysphagia), may be present as well.
- Decreased PO intake and pain on swallowing result in profound weight loss, easy fatigability, and weakness.
- With advanced disease, the patient will appear cachectic; supraclavicular lymphadenopathy may be present, as may signs of distant metastasis. May develop symptoms depending on local invasion (stridor, coughing, aspiration pneumonia, hemoptysis, vocal cord/recurrent laryngeal nerve paralysis).

EXAM TIP

Dysphagia does not usually develop until > 60% of the esophageal lumen is obstructed.

Diagnosis

- Population screening is untenable due to relatively low incidence, often absent early symptoms, rarity of a genetic cause.
- Asymptomatic patients are occasionally identified by surveillance endoscopy, especially patients with Barrett's esophagus.
- Barium esophagram is the initial diagnostic test—may show stricture, ulceration, or mass.
- EGD is useful to both visualize the mass and to retrieve specimens for biopsy.
  - Majority of adenocarcinoma is found in the distal esophagus; most SCCs are found in the middle and lower third. The cervical esophagus is an uncommon site for disease.
- CT scan of the thorax, abdomen, and pelvis is useful to define the extent of disease and thereby determine appropriate treatment.
- Endoscopic ultrasound (EUS) is useful to measure the depth of tumor invasion and presence of lymphadenopathy for preoperative staging and surgical planning.
- Positron emission tomographic (PET) scan for lymphatic spread.
- Staging according to TNM (tumor, node, metastasis) classification.

treAtment

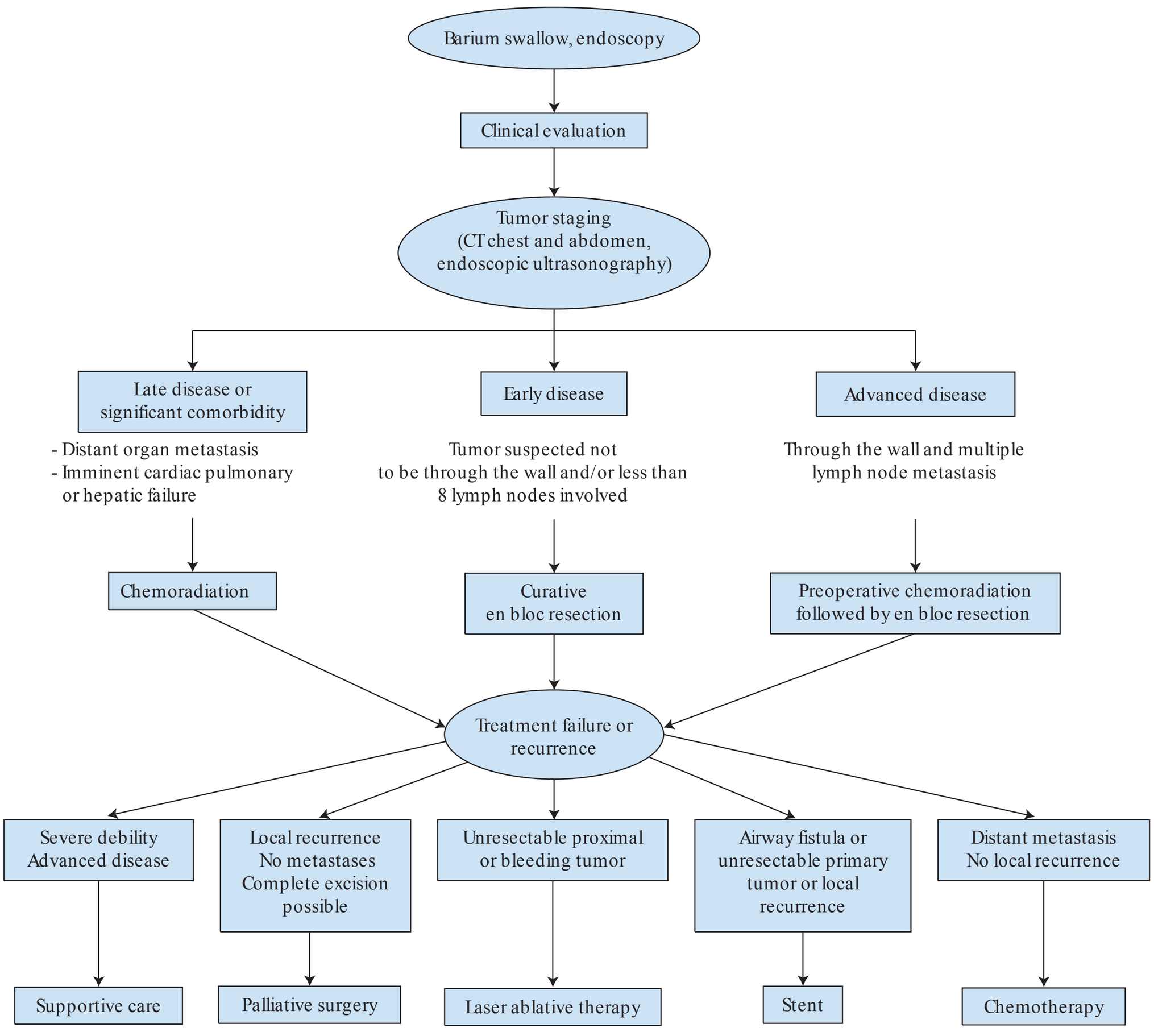
Surgical therapy vs. palliative surgical therapy vs. nonsurgical palliation (see Figure 7-4).

- Management of disease limited to the esophagus:
  - Surgical resection (right thoracic or transhiatal approach, gastric pull-up, or colonic interposition can be used to reconstruct the GI tract).
  - Radiotherapy for avoidance of perioperative morbidity and mortality (can shrink tumor but may predispose to local complications and not palliate dysphagia and odynophagia).
  - Pre/postoperative chemotherapy.
  - Combination therapy of these three modalities is becoming increasingly common.
- Treatment for advanced stage IV disease: Chemotherapy to promote tumor shrinkage and palliate symptoms; poor survival rate nonetheless.



WARD TIP

The 5-year survival rate for esophageal carcinoma is ~5%.



FIgu Re 7-4. suggested global algorithm for the management of carcinoma of the esophagus. CT = computed tomography.
(Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Figure 25-70.)

- Postoperative complications are common and include fistulae or abscesses and respiratory complications.
- Other options include endoscopic laser therapy, endoscopic dilatation and stent placement, or placement of a gastrostomy or jejunostomy.

Miscellaneous Esophageal Disorders

- Schatzki's ring: A thin, submucosal circumferential ring in the lower esophagus often associated with a hiatal hernia. Some believe it to be congenital, others due to infolding of redundant esophageal mucosa, and others due to stricture result from inflammation from chronic reflux.
  - Symptoms include brief episodes of dysphagia during hurried ingestion of solid foods.
  - Treatment ranges from dilatation +/- antireflux measures to incision of the ring and excision.
- Plummer–Vinson syndrome (Patterson–Kelly syndrome): An uncommon clinical syndrome characterized by dysphagia, atrophic oral mucosa, spoon-shaped and brittle fingernails, and chronic iron deficiency anemia. More common in perimenopausal women of Scandinavian origin.
  - An esophageal web, which is usually the cause of dysphagia was often thought to be a main component of the syndrome, but evidence has shown that it develops as a response to ingesting ferrous sulfate for the treatment of the anemia. Ferrous sulfate has been known to cause esophageal injury.
  - The web is usually below the cricopharyngeus muscle. Treatment consists of dilatation and iron therapy.
- "Differentiating Esophageal Ulcers"
  - CMV ulcers are typically linear, while HSV ulcers are typically punched out and deep

See Pediatric Surgery chapter for discussion of esophageal embryology and tracheoesophageal fistulas.

HIGH-YIELD FACTS IN

The STomach

Anatomy 108
- Blood Supply 108
- Innervation 108
- Histology and physiology 108

Peptic Ulcer Disease (PUD) 109
- epidemiology 109
- Complications 109

Duodenal Ulcer (DU) 109
- pathophysiology 109
- etiology 109
- Clinical Features 110
- diagnosis 110
- treatment 110

Gastric Ulcer (GU) 111
- pathophysiology 111
- etiology 112
- Classification 112
- Signs and Symptoms 112
- diagnosis 112
- treatment 112

Special Gastric Ulcers 113

Gastritis 113
- definition 113
- etiology, Signs, and Symptoms 113
- diagnosis 113
- treatment 113
- Complications 114

Postgastrectomy Complications 114
- postvagotomy diarrhea 114
- dumping Syndrome 114
- alkaline reflux gastritis 114
- afferent loop Syndrome 114
- nutritional deficiencies 114

Gastric Outlet Obstruction 115
- Common Causes 115
- Symptoms 115
- diagnosis 115
- treatment 115

Gastrointestinal Hemorrhage 115
- upper gI Hemorrhage 115

Bariatric Surgery 116
- Indication 116
- types 116

Malignant Tumors 117
- adenocarcinoma 117
- gastric lymphoma 120
- gastrointestinal Stromal tumor (gISt) 120

Benign Lesions 121
- Benign tumors/adenomatous polyps 121
- ménétrier's disease 121
- dieulafoy's lesion 121
- gastric Volvulus 122

OR TIP

Innervation of Stomach:
La RP
Left **a** nterior
Right **P**osterior

WARD TIP

Causes of Vitamin B_{12} deficiency:

- Gastrectomy: Loss of intrinsic factor-secreting tissue.
- Disease or resection of terminal ileum: Causing malabsorption of B_{12}.
- Pernicious anemia: Autoimmune destruction of parietal cells.
- Insufficient dietary intake (B_{12} is found in most foods of animal origin).

Anatomy

See Figure 8-1.

Blood Supply

- Greater curvature: Right and left gastroepiploic arteries.
- Lesser curvature: Right and left gastric arteries.
- Pylorus: Gastroduodenal artery.
- Fundus: Short gastric arteries.

Innervation

See Figure 8-2.

- Anterior gastric wall: Left vagus nerve (gives branch to liver).
- Posterior gastric wall: Right vagus nerve (gives celiac branch and the "criminal nerve of Grassi").
- Gastroduodenal pain: Sensation via sympathetic afferents from level T5 (below nipple line) to T10 (umbilicus).

Histology and Physiology

- Proximal cardiac glands: Secrete mucus.
- Fundus and body:
  - Parietal (oxyntic) cells: Secrete hydrochloride (HCl), accounting for the acidic pH of stomach; secrete intrinsic factor for absorption of vitamin B_{12} in the terminal ileum.
  - Chief (peptic) cells: Secrete pepsinogen, a proenzyme activated by gastric HCl to form pepsin, which digests protein.

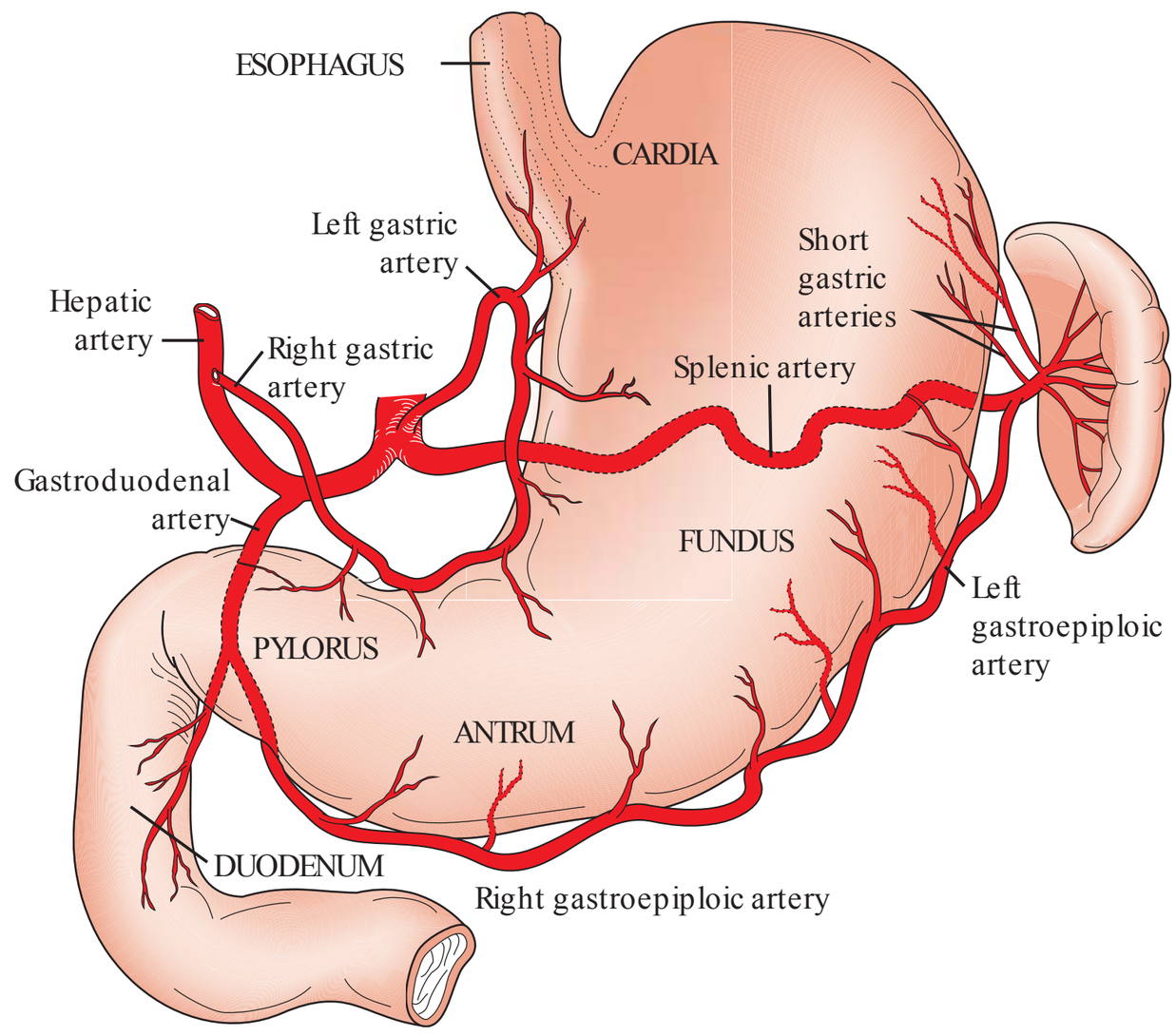


FIGURE 8-1. Anatomy of the stomach.

- Antrum: G cells—secrete gastrin, which stimulates gastric acid secretion, pepsin secretion, and mucosal growth of the gastrointestinal (GI) tract (trophic action).
- Acid secretion by parietal cells is stimulated by the vagus nerve (acetylcholine via muscarinic M3 receptors), histamine (via H_2 receptors), and gastrin (via gastrin receptors); the final common pathway is through the proton pump (H^+/K^+ ATPase).
- Gastrin release is stimulated by gastrin-releasing peptide (GRP) and the presence of digested protein products (amino acids) in the stomach; it is inhibited by somatostatin and low antral pH (< 2.5).
- Gastric mucosal barrier (protective gel layer) is enhanced by prostaglandin E (PGE) and damaged by nonsteroidal anti-inflammatory drugs (NSAIDs).
- Gastric bicarbonate secretion into the mucous gel is inhibited by NSAIDs, acetazolamide, alpha blockers, and alcohol.

Peptic Ulcer Disease (PUD)

PUD is classified by location; most commonly duodenal ulcers (DUs) vs. gastric ulcers (GUs).

epIdemIology

- Environmental factors: Helicobacter pylori infection, NSAID use, smoking.
- Other risk factors: Family history of ulcers, Zollinger–Ellison (gastrinoma), corticosteroids (high dose and/or prolonged course).
- Ulcer incidence increases with age for both GU and DU; DU emerges two decades earlier than GU, particularly in males.

ComplICatIonS

- Bleeding is the most common complication (20% incidence): Dizziness, syncope, hematemesis, melena.
- Perforation (7% incidence): Sudden, severe midepigastric pain radiating to right shoulder, peritoneal signs, free peritoneal air.
- Obstruction: Due to scarring and edema; early satiety, anorexia, vomiting, weight loss.

Duodenal Ulcer (DU)

pathopHySIology

Increased acid production (in contrast to GUs); also, H. pylori infection may weaken mucosal defenses.

etIology

- H. pylori: A bacterium that produces urease, which breaks down the protective mucous lining of the stomach; 70–90% of patients with PUD have H. pylori infection.
- NSAIDs/Steroids: Inhibit production of PGE, which stimulates mucosal barrier production.

WARD TIP

Typical scenario: A patient with known PUD presents with sudden onset of severe epigastric pain. Physical exam reveals guarding and rebound tenderness. Think: Perforation.

WARD TIP

alarm symptoms that indicate need for esophagogastroduodenoscopy (EGD):

- Weight loss
- Recurrent vomiting
- Dysphagia
- Bleeding
- Anemia

EXAM TIP

Typical scenario: A 52-year-old woman presents due to 3 months of early satiety, weight loss, and nonbilious vomiting. Think: Gastric outlet obstruction.

WARD TIP

Zollinger–Ellison syndrome accounts for 0.1–1% of patients with ulcer, but over 90% of patients with ZE have PUD (can see jejunal ulcers).

WARD TIP

H. pylori may colonize 50% of the global population—infection does not necessitate disease.

- Zollinger–Ellison (ZE) syndrome: Gastrinoma (gastrin-secreting tumor in or near the pancreas; two thirds are malignant); 20% of ZE patients have associated multiple endocrine neoplasia type 1 (MEN-1: parathyroid hyperplasia, pancreatic islet tumors, pituitary tumors); diarrhea is common.
- In the instance of a patient with recurrent duodenal ulcers not responding to H. pylori treatment, think Zollinger–Ellison syndrome, and check for concomitant pituitary and parathyroid problems (MEN 1).

EXAM TIP

Typical scenario: A 33-year-old female smoker presents with burning epigastric pain that is improved after eating a meal. Think: Duodenal ulcer.

Clinical Features

- Burning, gnawing epigastric pain that occurs with an empty stomach and is relieved by food or antacids (in contrast to GUs).
- Night time awakening (when stomach empties).
- Nausea, vomiting.
- Associated with blood type O.

diagnosis

- DU: Endoscopy; however, most symptomatic cases of DU are easily diagnosed clinically.
- H. pylori:
  - Endoscopy with biopsy—allows culture and sensitivity for H. pylori (organism is notoriously hard to culture—multiple specimens required during biopsy).
  - Serology: Anti–H. pylori immunoglobulin G (IgG) indicates current or prior infection.
- Urease breath test: $C^{13/14}$ labeled urea is ingested. If gastric urease is present, the carbon isotope can be detected as CO_2 isotopes in the breath.
- ZE: A fasting serum gastrin level > 1,000 pg/mL is pathognomonic for gastrinoma. Secretin stimulation test: Secretin (a gastrin inhibitor) is delivered parenterally and its effect on gastrin secretion is measured. In ZE syndrome, there is a paradoxical astronomic rise in serum gastrin.

EXAM TIP

Most common location for DU: Posterior duodenal wall within 2 cm of pylorus.

treatment

medical

- Risk modification:
  - Discontinue NSAIDs, steroids, smoking.
  - Prostaglandin analogues (e.g., misoprostol).
- Acid reduction:
  - Proton pump inhibitor (PPI): Omeprazole, lansoprazole, pantoprazole; 90% cure rate after 4 weeks.
  - H_2 blockers (cimetidine, ranitidine, famotidine, nizatidine): 85–95% cure rate after 8 weeks.
  - Antacids: Over the counter, good for occasional use for all causes of dyspepsia.
- Eradication of H. pylori:
  - Triple therapy (2-week regimen with bid dosing): PPI + amoxicillin + clarithromycin.
  - If patient is penicillin allergic, can substitute metronidazole for amoxicillin.
  - If patient fails one course of therapy, can try an alternate regimen using a different combination of drugs or quadruple therapy (2-week regimen of PPI + bismuth + tetracycline + metronidazole).

WARD TIP

Triple therapy has 70–85% eradication rate.
Quadruple therapy has 75–90% eradication rate.

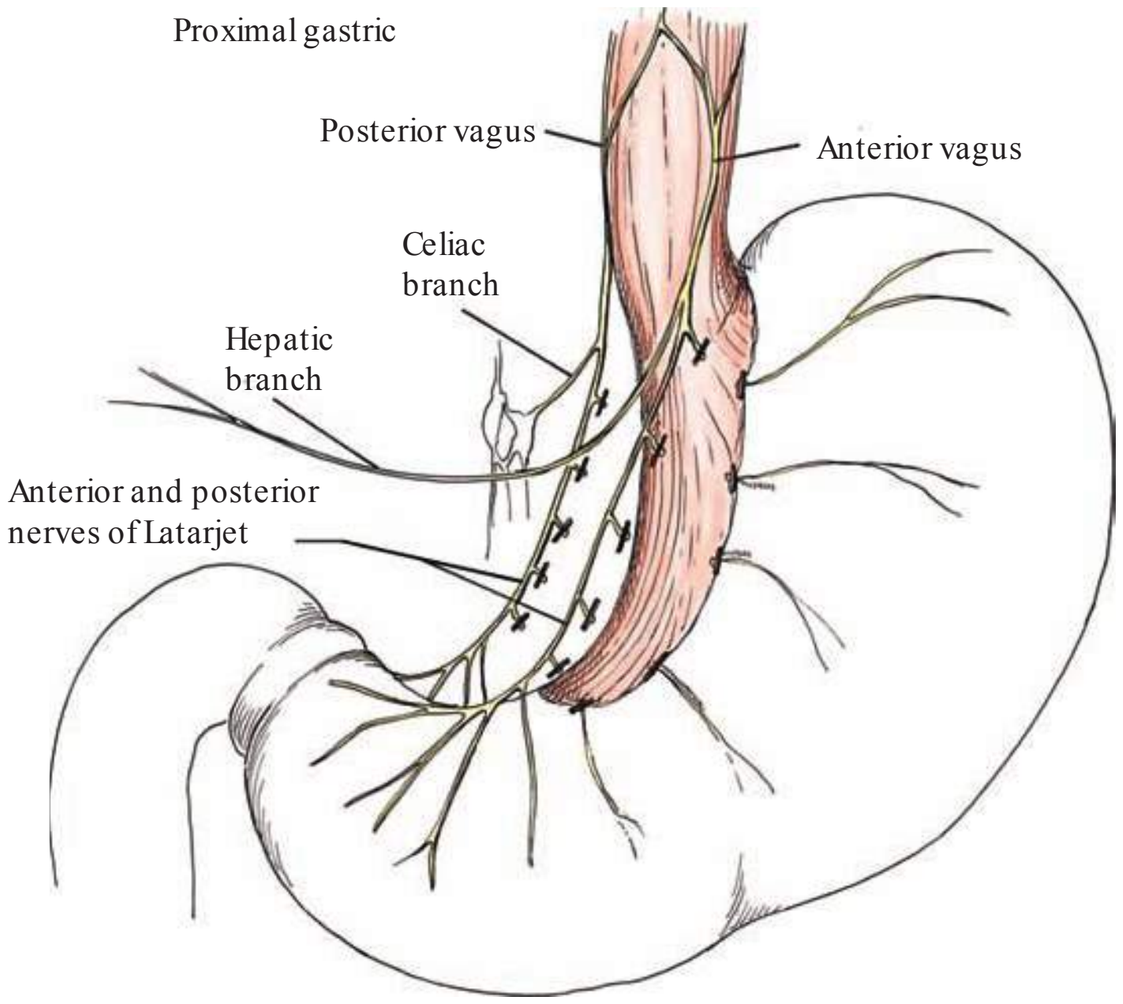


FIGURE 8-2. highly selective vagotomy. (Reproduced, with permission, from Zollinger RM Jr, Zollinger RM Sr. Zollinger's Atlas of Surgical Operations. 8th ed. New York: McGraw-Hill; 2003: 45.)

Surgical

- Since the advent of highly effective medical therapy, elective surgery for PUD is quite rare.
- Surgery is indicated when ulcer is refractory to 12 weeks of medical treatment or if hemorrhage, obstruction, or perforation is present.
- Truncal vagotomy and selective vagotomy are not commonly performed anymore due to associated morbidity (high rate of dumping syndrome) despite good protection against recurrence.
- Procedure of choice is highly selective vagotomy (parietal cell vagotomy, proximal gastric vagotomy) (see Figure 8-2).
- Individual branches of the anterior and posterior nerves of Latarjet in the gastrohepatic ligament going to the lesser curvature of the stomach are divided from a point 6 cm proximal from the pylorus to a point 6 cm proximal to the esophagogastric junction. The terminal branches to the pylorus and antrum are spared, preserving pyloroantral function and thus obviating the need for gastric drainage.
- Preferred due to its lowest rate of dumping; however, it does have the highest rate of recurrence.
- Recurrence depends on site of ulcer preop: Prepyloric ulcers have the highest recurrence rate at 30%. Least recurrence rate is with vagotomy + antrectomy.
- Laparoscopic option: A posterior truncal vagotomy coupled with an anterior seromyotomy is being done laparoscopically.
- For ZE: The tumor is resected. Occasionally, when focus of tumor cannot be found, a total gastrectomy may be considered in severe cases refractory to medical management.

Gastric Ulcer (GU)

pathophysiology

- Decreased protection against acid; acid production may not be elevated (in contrast to DUs).

EXAM TIP

Common complications specific to surgery for peptic ulcer disease:
Ga me Pa D
Gallstones
a fferent loop syndrome
marginal ulcer
efferent loop obstruction
Postvagotomy diarrhea (most common)
a lkaline reflux gastritis
Dumping syndrome

WARD TIP

Gastric ulcers can even occur with achlorhydria.

WARD TIP

Smoking is a risk factor for GU.

WARD TIP

Signs of duodenal perforation:
Bleeding from the **B**ack (posterior duodenal erosion/perforation, involving gastroduodenal artery).
Free **a** ir from **a** nterior duodenal perforation.
Note: Anterior perforation is more common than posterior.

EXAM TIP

Typical scenario: A 45-year-old Japanese male smoker presents with weight loss and epigastric pain exacerbated by eating. Think: Gastric ulcer.

- Can be caused by reflux of duodenal contents (pyloric sphincter dysfunction) and decreased mucus and bicarbonate production.

Etiology

- NSAIDs and steroids inhibit production of prostaglandins (PGE stimulates production of protective gastric mucosal barrier).
- H. pylori produces urease, which breaks down the gastric mucosal barrier.

Classification

- Location determines classification and is important in determining treatment (Table 8-1).
- Aid to memory: One is Less, Two has Two, Three is Pre, Four is by the Door.

Signs and Symptoms

- Burning, gnawing epigastric pain that occurs with anything in the stomach; pain is worst after eating (in contrast to DU).
- Anorexia/weight loss.
- Vomiting.
- Associated with blood type A.

diagnosis

- Double contrast barium swallow.
- Endoscopy.
- All GUs are biopsied—3% are associated with gastric cancer.

treatment

- Medical options: Same as for DUs.
- Surgical options: See Tables 8-1 and 8-2.

TABLE 8-1. Classification, Pathogenesis, and Surgical Treatment Options for Gastric Ulcer

Type	Location of Ulcer	Pathogenesis	Surgical Treatment
I	Most common; near angularis incisura on lesser curvature	Normal or decreased acid secretion; decreased mucosal defense	Distal gastrectomy with ulcer excision
II	Associated with DU (active or quiescent)	Normal or increased acid secretion	Antrectomy with truncal vagotomy and ulcer excision
III	Prepyloric		
IV	Near gastroesophageal junction	Normal or subnormal acid secretion; decreased mucosal defense	Distal gastrectomy with ulcer excision and esophagogastrojejunostomy

TABLE 8-2. Surgical options in the Treatment of Duodenal and Gastric Ulcer Disease

Indication	Duodenal	Gastric
Bleeding	1. Oversew[a] 2. Oversew, V+ D 3. V+ A	1. Oversew and biopsy[a] 2. Oversew, biopsy, V+ D 3. Distal gastrectomy[b]
Perforation	1. Patch[a] 2. Patch, HSV[b] 3. Patch, V+ D	1. Biopsy and patch[a] 2. Wedge excision, V+ D 3. Distal gastrectomy[b]
Obstruction	1. HSV+ GJ 2. V+ A	1. Biopsy; HSV+ GJ 2. Distal gastrectomy[b]
Intractability/nonhealing	1. HSV[b] 2. V+ D 3. V+ A	1. HSV and wedge excision 2. Distal gastrectomy[a]

[a]Unless the patient is in shock or moribund, a definitive procedure should be considered.

[b]Operation of choice in low-risk patient.

GJ = gastrojejunostomy; HSV = highly selective vagotomy; V+ A = vagotomy and antrectomy; V+ D = vagotomy and drainage.

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 967.

Special Gastric Ulcers

- Curling's ulcers: Gastric stress ulcers in patients with severe burns.
- Cushing's ulcers: Gastric stress ulcer related to severe central nervous system (CNS) damage.

Gastritis

Definition

Acute or chronic inflammation of the stomach lining.

Etiology, Signs, and Symptoms

Similar to PUD; endoscopy needed to differentiate.

Diagnosis

Diagnosis is made by endoscopy.

Treatment

Same as medical treatment of GUs.

WARD TIP

- Burnt paper CURLS.
- Cush**h**ing's ulcer (think: **h ead** = CNS trauma/tumor).

WARD TIP

- Always biopsy a gastric ulcer to rule out cancer. Duodenal ulcers are almost always associated with H. pylori and don't need to be biopsied.

WARD TIP

Etiologies of gastritis:
GNa Sh ING
Gastric reflux (bile or pancreatic secretions)
Nicotine
alcohol
Stress
helicobacter pylori and other infections
Ischemia
NSAIDs
Glucocorticoids (long-term use)

Complications

Chronic gastritis leads to:

- Gastric atrophy.
- Gastric metaplasia.
- Pernicious anemia (decreased production of intrinsic factor from gastric parietal cells due to idiopathic atrophy of the gastric mucosa and subsequent malabsorption of vitamin B_{12}).

EXAM TIP

Cimetidine is a p450 inhibitor, and therefore prolongs the action of drugs cleared by this system.

Postgastrectomy Complications

Postvagotomy Diarrhea

Dumping Syndromes

EXAM TIP

Two types of chronic gastritis and their associations:
Type **a** (fundal)—pernicious **a**nemia, parietal cell **a**ntibodies, **a**chlorhydria, **a**utoimmune disease.
Type **B** (antral)—**B**ug (H. pylori infection in almost all cases).

- Complication of gastric surgery thought to result from unregulated movement of gastric contents from stomach to small intestine.
- Symptoms typically occur 5–15 minutes postprandially (early dumping syndrome) due to high osmolar load reaching the small intestine or 2–4 hours postprandially (late dumping syndrome) due to hypoglycemia.
- Nausea, vomiting, belching, diarrhea, tachycardia, palpitations, flushing, diaphoresis, dizziness, syncope.
- Treated by dietary modification: Small, multiple low-carbohydrate/fat meals; avoid excessive liquid intake.
- Severe cases (1%) that do not respond to dietary modifications can be treated with octreotide (synthetic somatostatin—helps delay gastric emptying time and transit through small intestine).

EXAM TIP

Typical scenario: A 58-year-old woman who is 6 days postop from a gastrojejunostomy for PUD presents with postprandial RUQ pain and nausea. She reports that vomiting relieves her suffering. Think: Afferent loop syndrome.

Alkaline Reflux Gastritis

- Diagnosis of exclusion after recurrent ulcer has been ruled out; nonspecific EGD and biopsy findings (edematous, inflamed gastric mucosa).
- Presents with postprandial pain and bilious vomiting.
- Medical treatment is difficult.
- Surgical management: Roux-en-Y gastrojejunostomy with a long (~50-cm) Roux limb. Bilious vomiting may improve, but symptoms (early satiety, bloating) may persist.

EXAM TIP

CAUSES:
mallory's **V**ices **G**ave (her) An Ulcer.
Mallory–Weiss tear
Varices
Gastritis
AV malformation
Ulcers

Afferent Loop Syndrome

- Obstruction of afferent limb following gastrojejunostomy (Bilroth II); two thirds present in postop week 1.
- **Symptoms:** Postprandial right upper quadrant (RUQ) pain, bilious vomiting, steatorrhea (with concomitant malabsorption of fats, B_{12}), anemia.
- **Diagnosis:** Afferent loop will be devoid of contrast of UGI series.
- **Treatment:** Endoscopic balloon dilatation or surgical revision of loop if that fails.

Nutritional Deficiencies

- Vitamin B_{12} deficiency anemia.
- Iron deficiency anemia.
- Osteoporosis (due to reduced calcium absorption).

Gastric Outlet Obstruction

Common Causes

- Malignant tumors of stomach and head of pancreas.
- Obstructing gastric or DUs.
- Usually with DU.
- Chronic ulcer causes secondary edema or scarring, which occludes lumen.

Symptoms

Early

- Early satiety.
- Gastric reflux.
- Abdominal distention.

Late

- Vomiting.
- Dehydration.
- Hypochloremic, hypokalemic metabolic alkalosis with paradoxical aciduria.
- Weight loss.

Diagnosis

Endsocopy or barium swallow x-ray.

Treatment

- Truncal vagotomy and pyloroplasty or gastrojejunostomy after 7 days of nasogastric (NG) decompression and antisecretory treatment.
- NG decompression is necessary to normalize the size of the dilated stomach.

WARD TIP

CONTRAST:
mallory–Weiss syndrome: Postemetic tears in gastric mucosa (near gastroesophageal junction).
Boerhaave syndrome: Postemetic esophageal rupture.

WARD TIP

Coffee grounds is the term used to describe old, brown digested blood found on gastric lavage. It usually indicates a source of bleeding proximal to the ligament of Treitz.

Gastrointestinal Hemorrhage

Upper GI Hemorrhage

Etiology

- Ulcer (peptic)
- Varices
- Gastritis
- Arteriovenous malformation
- Mallory–Weiss tear

Signs and Symptoms

- Hematemesis (bright red or coffee grounds).
- Hypotension.
- Tachycardia.

WARD TIP

A bleeding scan detects active bleeding by infusing technetium-labeled autologous red blood cells (^{99m}Tc-labeled RBCs) and watching for their collection in the GI tract. It can be completed in ~1 hour and can detect bleeds as slow as 0.1 mL/min, but location specificity is only 60–70%. CT angiography is faster and detects bleeds up to 0.5 mL/min.

WARD TIP

Boerhaave syndrome: Postemetic esophageal transmural rupture (left sided, 2–3 cm above GEJ)

EXAM TIP

- In setting of gastric varices, look for splenic vein thromboses secondary to pancreatitis, pancreatic malignancy, or other abdominal malignancy

EXAM TIP

- Gastric lavage results:
  - Bloody: (+) upper GI source of bleed.
  - Bilious: (-) upper GI source of bleed.
  - Clear: equivocal; NG tube should be inserted deeper and test repeated.

WARD TIP

Risk of ulcers rebleeding:
- In-hospital: One third.
- GUs are three times more likely to rebleed than DUs.

WARD TIP

Medical conditions commonly associated with morbid obesity: Diabetes mellitus, coronary artery disease, hypertension, sleep apnea, arthritis, sex hormone abnormalities, breast cancer, colon cancer.

- Bleeding that produces 60 cc of blood or more will produce black, tarry stool (melena).
- Very brisk upper GI bleeds can be associated with bright red blood per rectum (hematochezia) and hypotension.

Diagnosis

- Gastric lavage with normal saline or free water to assess severity of bleeding (old vs. new blood).
- Rectal exam.
- Complete blood count (CBC).
- Endoscopy.
- Bleeding scan.
- Arteriography.

Treatment

- Depends on etiology and severity.
- Bleeding varices are ligated, or sclerosed via endoscopy (see Hepatobiliary System chapter).
- Most Mallory–Weiss tears resolve spontaneously.
- For severe bleeds:
  - Intravenous fluids and blood products as needed.
  - Somatostatin (inhibits gastric, intestinal, and biliary motility, decreases visceral blood flow).
  - Consider balloon tamponade for esophageal varices.
- Surgery:
  - About 5% of the time, upper GI bleeding cannot be controlled via endoscopic or other methods and emergent laparotomy will be necessary.
  - For DUs, a longitudinal incision is made across the pylorus and proximal duodenum. Bleeding is controlled by undersewing the vessel on either side of the hemorrhage.

Bariatric Surgery

Indication

BMI (body mass index) = weight in kg/(height in M)^2:
- BMI > 35 with comorbidity (e.g., hypertension, diabetes mellitus).
- BMI ≥ 40 with or without comorbidity.

Prerequisite: Participation in supervised dietary program without success.

types

- Restrictive: Reduction of the quantity of food intake.
  - Vertical banded gastroplasty (VBG): Partitioning of the stomach into a small proximal pouch (<20 mL) and a more distal one (see Figure 8-3).
  - Sleeve gastric resection.
  - Laparoscopic adjustable band placement (Figure 8-4).
- Malabsorptive: Limit nutrient absorption by bypassing duodenum and small intestine.
  - Biliopancreatic diversion with or without duodenal switch (Figure 8-5).
  - Roux-en-Y gastric bypass (also has a restrictive component; see Figure 8-6).

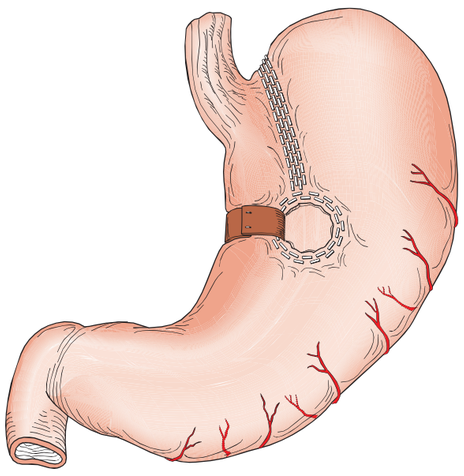


FIGURE 8-3. Vertical banded gastroplasty.

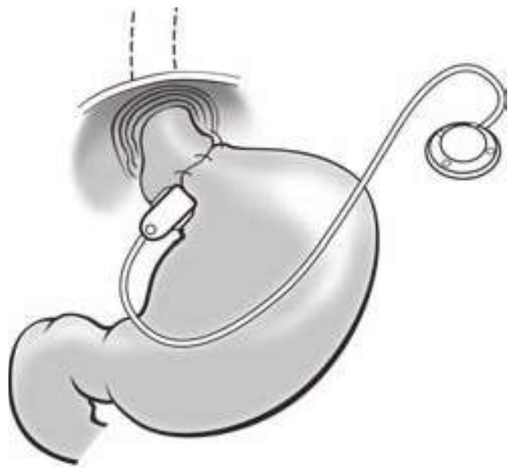


FIGURE 8-4. Adjustable gastric banding with gastropexy. (Reproduced, with permission, from Miller K. Laparoscopic restrictive procedures: adjustable gastric banding. In Pitombo C, Jones KB Jr, Higa KD, Pareja JC, eds. Obesity Surgery: Principles and Practice. New York: McGraw-Hill; 2008: 170.)

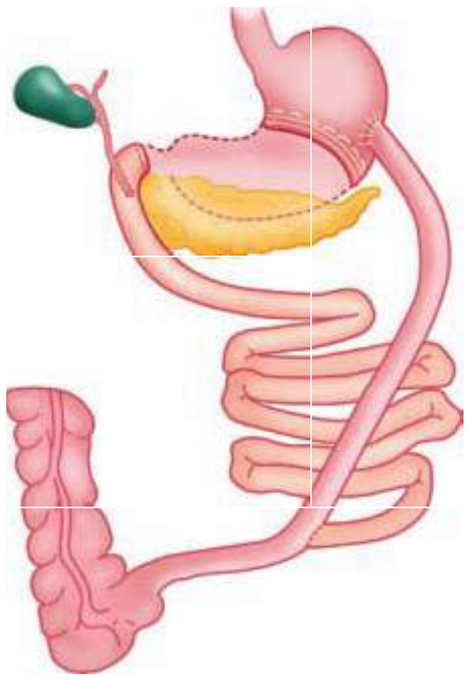


FIGURE 8-5. Biliopancreatic diversion procedure before the gallbladder is removed. (Reproduced, with permission, from Jacobs DO, Robinson MK. Morbid obesity and operations for morbid obesity. In Zinner MJ, Ashley SW, eds. Maingot's Abdominal Operations. 11th ed. New York: McGraw-Hill; 2007: 470.)



FIGURE 8-6. Roux-en-Y gastric bypass.

Malignant Tumors

Most common stomach malignancies are Adenocarcinoma (95%), Lymphoma (4%), and GIST (1%).

adenoCarCinoma

epidemiology

- In general, gastric cancer is a disease of the elderly (age > 60), men > women, blacks > whites.
- Adenocarcinoma comprises 95% of malignant gastric cancer.
- Leading cause of cancer-related death in Japan.

Risk Factors

- Familial adenomatous polyposis.
- Chronic atrophic gastritis.
- H. pylori infection (6× increased risk).
- Post-partial gastrectomy (15+ years).
- Pernicious anemia.

- Diet (foods high in nitrites—preserved, smoked, cured).
- Cigarette smoking.
  - Epstein–Barr virus infection.

Pathology

- Polypoid: 25–50%, no substantial necrosis or ulceration.
- Ulcerative: 25–50%, sharp margins.
- Superficial spreading: 3–10%, involves mucosa and submucosa only, best prognosis.
- Linitis plastica: 7–10%, "leather bottle" type, involves all layers, extremely poor prognosis.

Signs and Symptoms

- Early: Mostly asymptomatic.
- Late: Anorexia/weight loss, nausea, vomiting, dysphagia, melena, hematemesis; pain is constant, nonradiating, exacerbated by food.
- Anemia—from blood loss, pernicious.
- Krukenberg's tumor—metastasis to ovaries.
- Blumer's shelf—metastasis to pelvic cul-de-sac, felt on digital rectal exam.
- Virchow's node—metastasis to lymph node palpable in left supraclavicular fossa.
- Sister Mary Joseph's nodule—periumbilical metastatic nodules.

Diagnosis and Preoperative Staging

- Upper GI endoscopy: Best method for diagnosis, allows for biopsy, definitive > 95% sensitivity and specificity.
- Upper GI series: With double contrast; 80–96% sensitivity, 90% specificity (operator dependent); excellent method in skilled hands.
- Abdominal and pelvic CT: With IV and oral contrast for preop staging to detect metastatic disease.
- Endoscopic ultrasound: For preop local assessment of tumor including depth of invasion and perigastric nodes.

Staging

See Table 8-3.

Treatment

- Radical subtotal gastrectomy can be curative in early disease confined to the superficial layers of the stomach (less than one third of all patients due to typical late presentation). Patients with proximal cancer may need total gastrectomy with Roux-en-Y reconstruction.
- Chemotherapy: Sometimes used palliatively for nonsurgical candidates; marginal benefit in adjuvant setting.

Prognosis

- Treatment is a major prognostic factor for gastric cancer—patients who are not resected have a poor prognosis.
- Location: Proximal gastric cancer has less favorable prognosis than distal lesions.
- Tumor markers: High preop serum levels of carcinoembryonic antigen (CEA) and CA 19-9 have been associated with less favorable outcomes.
- Other factors: Histologic grade, regional lymphatic spread.

TABLE 8-3. Gastric adenocarcinoma TNM classification

TUMOR (T)	
Tx	Tumor cannot be assessed
T0	No evidence of tumor
Tis	Carcinoma in situ (no invasion)
T1	Tumor invades lamina propria, muscularis mucosae, or submucosa
T2	Tumor invades muscularis propria
T3	Tumor penetrates subserosal tissue without invasion of serosa or adjacent structures
T4	Tumor invades serosa (4a) or adjacent structures (4b)

LYMPH NODES (N)	
Nx	Lymph nodes cannot be assessed
N0	No lymph node metastasis
N1	Metastasis in 1–2 regional lymph nodes
N2	Metastasis in 3–6 regional lymph nodes
N3	Metastasis in 7 or more regional lymph nodes

DISTANT METASTASIS	
M0	No distant metastasis
M1	Distant metastasis

STAGE	**T**	**N**	**M**
0	Tis	N0	M0
IA	T1	N0	M0
IB	T2	N0	M0
	T1	N1	M0
IIA	T3	N0	M0
	T2	N1	M0
	T1	N2	M0
IIB	T4a	N0	M0
	T3	N1	M0
	T2	N2	M0
	T1	N3	M0
IIIA	T4a	N1	M0
	T3	N2	M0
	T2	N3	M0
IIIB	T4b	N0	M0
	T4b	N1	M0
	T4a	N2	M0
	T3	N3	M0

(continues)

Table 8-3. Gastric adenocarcinoma TNm classification (continued)

Stage	T	n	m
IIIC	T4b	N2	M0
	T4b	N3	M0
	T4a	N3	M0
IV	Any T	Any N	M1

gastric lymphoma

- Second most common malignant gastric cancer.
- Stomach is most common site for primary GI lymphoma (majority are B-cell non-Hodgkin's type) but lymphoma comprise only 4% of all gastric tumors.
- Increased risk with H. pylori infection.

Signs and Symptoms

Nonspecific; include abdominal discomfort, nausea, vomiting, anorexia, weight loss, and hemorrhage; occult bleeding and anemia (50% of patients).

Diagnosis

- Made by endoscopic biopsy, not readily distinguishable from adenocarcinoma by simple inspection.
- Bone marrow aspiration and gallium bone scans can diagnose metastases.

Treatment

- MALT (low grade)—treat H. pylori.
- MALT (high grade) or non-MALT—radiation/chemo ± surgical resection.
- Resection reserved for patients with bleeding or perforation.

Prognosis

Poor prognostic factors include:

- Involvement of the lesser curvature of the stomach.
- Large tumor size.
- Advanced stage.

gastrointestinal stromal tumor (gIst)

- Mesenchymal (smooth muscle/interstitial cells of Cajal) tumors arising from the gastric stroma; submucosal and slow growing.
- Stomach is the most common site.
- Variable histology—from spindle cell tumors to epithelioid to pleomorphic.
- Approximately 95% of GISTs have c-kit (CD117) expression, leading to a constitutively active receptor tyrosine kinase.
- All GISTs are regarded as malignant.
- Treated by surgical resection and Gleevec (imatinib) (inhibits the tyrosine kinase receptor).
- Prognosis depends on completeness of resection, presence of metastases, and the mitotic index.

Benign Lesions

Benign tumors/adenomatous polyps

- Account for 10–20% of all gastric polyps.
- Are the only ones with any real malignant potential; others are mostly asymptomatic and uncommon.
- Biopsy lesions > 5 mm to check for neoplasia.

ménétrier's disease

Definition

- Hypertrophic gastropathy (enlarged, tortuous gastric rugae).
- Protein-losing enteropathy.
- Mucosal thickening secondary to hyperplasia of glandular cells replacing chief and parietal cells.
- Low-grade inflammatory infiltrate—not a form of gastritis.

Signs and Symptoms

- Most common: Middle-aged man who presents with epigastric pain, weight loss, diarrhea, hypoproteinemia.
- Less common: Nausea, vomiting, anorexia, occult GI bleed.
- Gastric acid secretion can be high, normal, or low.

Diagnosis

- Endoscopy with deep mucosal biopsy is definitive.
- Barium swallow will reveal large gastric folds and thickened rugae.

Treatment

- Anticholinergics, H_2 blockers to reduce protein loss.
- High-protein diet.
- Treatment of ulcers/cancer if present and eradication of H. pylori.
- Severe disease may require gastrectomy.

WARD TIP

Ménétrier's can look like gastric cancer on barium study.

WARD TIP

Monitor closely as there may be increased incidence of gastric cancer (adenocarcinoma).

dieulafoy's lesion

Definition

Mucosal end artery that causes pressure necrosis and erodes into stomach and ruptures.

Symptoms

Massive, recurrent painless hematemesis.

Diagnosis

Upper GI endoscopy.

Treatment

- Endoscopic sclerosing therapy or electrocoagulation.
- Wedge resection.

gaStr ICvol vul uS

Def nition

Torsion/twisting of stomach typically along long axis. Often associated with paraesophageal hernia. May be acute, but most often chronic.

Symptoms

Brochardt's triad:

- Intermittent severe epigastric pain and distention.
- Inability to vomit.
- Difficult passage of NG tube.

Diagnosis

Upper GI contrast study.

Treatment

- Surgical repair of accompanying hernia.
- Gastropexy—fixes stomach to anterior abdominal wall.
- Gastric resection if there is necrosis.

HIGH-YIELD FACTS IN

Small Bowel

Introduction 124

Gastrointestinal (GI) Embryology 124
- Fourth Week 124
- Fifth Week 124
- tenth Week 124

Gross Anatomy 124
- General 124
- Duodenum 124
- Duodenal Blood Supply 125
- Jejunum and Ileum 125
- lymphatics 125
- innervation 125
- peristalsis 126

GI Hormones 126
- absorption overview 126
- intestinal immune Function 126

Disorders of the Small Bowel 126
- Small Bowel obstruction (SBo) 126
- crohn's Disease 131
- Benign neoplasms of Small intestine 134
- malignant neoplasms of Small intestine 134
- carcinoid 137
- Fistula 138
- mesenteric ischemia 139
- acute mesenteric ischemia (ami) 139
- chronic mesenteric ischemia (cmi) 140
- Short Bowel Syndrome 141

EXAM TIP

All of the small intestine is derived from the midgut except for the proximal duodenum, which is derived from the foregut. The junction between the foregut and midgut is immediately distal to the opening of the common bile duct.

Introduction

The small bowel is the principal site for the absorption and digestion of nutrients, as well as for the maintenance of fluid homeostasis in the gastrointestinal system. It also serves as a major component of both the endocrine and immune systems.

Gastrointestinal (GI) Embryology

Fourth Week

The primitive gut tube, formed from the endoderm, begins to develop into the foregut, midgut, and hindgut.

- Endoderm becomes intestinal epithelium and glands.
- Mesoderm becomes connective tissue, muscle, and wall of intestine.

EXAM TIP

Initially, the primitive gut tube communicates with the yolk sac. This communication narrows by the sixth week to form the vitelline duct. If the vitelline duct fails to obliterate by the end of gestation, it persists as a Meckel's diverticulum seen in 2% of the population.

FiFth Week

- Intestine elongates and midgut loop herniates through umbilical ring.
- Midgut loop continues to lengthen extracoelomically until approximately week 10.

tenth Week

Midgut loop rotates 270° counterclockwise and returns back into the abdominal cavity.

EXAM TIP

The midgut loop rotates a total of 270° around the axis of the superior mesenteric artery (SMA) before it reaches its final fixed position in the abdomen.

Gross Anatomy

General

- Total length: 5–10 m (average 6 m).
- Consists of three parts: Duodenum, jejunum, ileum
- Aside from the first 2 cm, the duodenum is a retroperitoneal structure, while the jejunum and ileum are intraperitoneal structures.

Duodenum

- Extends from the pylorus to the duodenojejunal junction.
- Consists of four parts:
  - Superior (first) part—duodenal bulb: 5 cm long; site of most ulcers.
  - Descending (second) part—10 cm long; curves around the head of pancreas.
  - Transverse (third) part—10 cm long; crosses anterior to aorta and inferior vena cava (IVC) and posterior to the SMA and superior mesenteric vein (SMV).
  - Ascending (fourth) part—5 cm long; ascends past left side of aorta, then curves anteriorly to meet with jejunum, forming the duodenojejunal junction, which is suspended by the ligament of Treitz.
- Duodenum ends and jejunum begins at the ligament of Treitz.

- Plicae circulares (transverse mucosal folds in the lumen of the small bowel) are more prominent in the proximal small bowel (duodenum and jejunum) than in the distal small intestine (ileum).

Duodenal Blood Supply

- Arterial supply:
  - Proximal (up to ampulla of Vater): Gastroduodenal artery (first branch of proper hepatic artery) bifurcates into the anterior and posterior superior pancreaticoduodenal arteries.
  - Distal (beyond ampulla of Vater): Inferior pancreaticoduodenal artery (first branch of SMA) bifurcates into the anterior and posterior inferior pancreaticoduodenal arteries.
- Venous drainage:
  - Anterior and posterior pancreaticoduodenal veins drain into the SMV, which joins the splenic vein behind the neck of the pancreas to form the portal vein.
  - Prepyloric vein of Mayo is landmark for pylorus.

Jejunum and Ileum

- No anatomical boundary between the two.
- Jejunum is the proximal 40% of small intestine distal to ligament of Treitz.
- Ileum is the distal 60% of small intestine.
- Combined length is 5–10 m (average 6 m).
- Mesentery tethers the jejunum and ileum to posterior abdominal wall.
- Arterial supply:
  - Both jejunum and ileum supplied by branches of SMA, which runs in the mesentery.
  - The arteries loop to form arcades that give rise to straight arteries—vasa recta.
- Venous drainage: The SMV drains both the jejunum and ileum.

Lymphatics

- Drainage: Bowel wall → mesenteric nodes → lymphatic vessels parallel the corresponding arteries → cisterna chyli (a retroperitoneal structure between the aorta and IVC) → thoracic duct (also between the aorta and IVC) → left subclavian vein.
- Participate in absorption of fat.

Innervation

Parasympathetic System

- Source: Fibers originate from vagus and celiac ganglia.
- Function: Enhances bowel secretion, motility, and other digestive processes.

Sympathetic System

- Source: Fibers originate from ganglion cells that reside in a plexus at the base of the SMA.
- Function: Opposes effects of parasympathetic system on bowel.

Enteric Nervous System

Consists of Meissner plexus at base of submucosa and Auerbach plexus between the inner circumferential and outer longitudinal layers of the muscle wall.

EXAM TIP

Various congenital anomalies are associated with either failure of midgut loop to rotate properly (i.e., malrotation) or failure of retraction of midgut loop back into the abdominal cavity (i.e., omphalocele and gastroschisis). (See Pediatric Surgery chapter for more details.)

EXAM TIP

- Ampulla of Vater/Major duodenal papilla, which is surrounded by sphincter of Oddi, is located in second portion of the duodenum.
- The junction between the foregut and midgut is the ligament of Treitz.

WARD TIP

Anterior ulcers tend to perforate, causing leakage of duodenal contents into the peritoneal cavity, leading to peritonitis. Ulcers that result in massive bleeding are posterior ulcers that have penetrated the gastroduodenal artery.

EXAM TIP

Typical scenario: A patient with a history of duodenal ulcers presents with massive GI bleeding. Upper GI endoscopy shows an ulcer that penetrates the posterior wall of the duodenal bulb (first part of the duodenum). What is the most likely vascular structure responsible for the bleeding? Think: The gastroduodenal artery courses behind the first part of the duodenum and is the most likely culprit.

EXAM TIP

Since the third part of the duodenum lies posterior to the SMA, compression of the SMA on the duodenum, as seen in SMA syndrome, can lead to small bowel obstruction (SBO). Although rare, this is most typically seen in thin patients who have lost the fat pad between the SMA and the duodenum, leading to recurrent symptoms of SBO. Treatment consists of weight gain/nutrition, or a surgical bypass procedure.

WARD TIP

Plicae circulares are helpful on abdominal x-rays, to differentiate between the small and large bowel (the colon has haustra, it does not have plicae circulares).

OR TIP

The entire small bowel is supplied by branches of the SMA except the proximal duodenum (which is supplied by branches from the celiac trunk).

OR TIP

How do you differentiate between jejunum and ileum upon gross inspection?

- Jejunum has larger diameter, thicker wall, and more prominent plicae circulares.
- The jejunum has few (1–2) arcades with long vasa recta, and the ileum has many arcades with short vasa recta.
- Ileum has fatty mesentery.

WARD TIP

Since > 93% of ingested fat is normally absorbed, > 6 g of fecal fat collected over a 24-hour time period in a diet with 100 g of lipid ingested per day would be defined as steatorrhea.

Peristalsis

- Intestinal contractions at a rate of 1–2 cm/sec.
- Main function is to move chyme through the intestine.

GI Hormones

See Table 9-1.

Absorption overview

See Table 9-2.

Intestinal immune Function

- Gut is largest immune organ in human body.
- Immunoglobulin A (IgA) is most prevalent type of immunoglobulin in lumen of GI tract, part of initial immune defense.
- Lymphoid nodules, mucosal lymphocytes, and isolated lymphoid follicles in appendix and mesenteric lymph nodes together constitute the mucosa-associated lymphoid tissue (MALT).

Disorders of the Small Bowel

Small Bowel obstruction (SBo)

Definition

- Cessation, impairment, or reversal of the physiologic transit of intestinal contents secondary to a mechanical or functional cause.
- Most common cause is adhesions from prior abdominal surgery (75%).

etiology

Mechanical

- Extrinsic (adhesion, hernia, cancer, abscess, congenital).
- Intraluminal (gallstone ileus, foreign body, intussusception).
- Intramural (Crohn's disease, lymphoma, radiation enteritis).

Functional (Paralytic ileus)

- Postoperative.
- Electrolyte abnormalities (e.g., hypokalemia).
- Peritonitis.
- Medications (opiates, anticholinergics).
- Hemoperitoneum/retroperitoneal hematoma.

Pathophysiology

- Gas and fluid begin to accumulate within lumen, proximal to site of obstruction.
- The bowel distends, intramural and intraluminal pressure rises.
- If intramural pressure exceeds the pressure in the microvasculature, then perfusion of the intestine is decreased, resulting in small bowel ischemia, and ultimately necrosis.
- Impairment of perfusion is termed strangulation.

Table 9-1. Hormones of the GI Tract

Hormone	Site of release	Action	Stimulated By	Inhibited By
Gastrin	Antrum	▪ Gastric acid secretion ▪ Cell growth	▪ Vagus ▪ Food in antrum ▪ Gastric distention ▪ Calcium	▪ Antral pH < 2.0 ▪ Somatostatin
Cholecystokinin (CCK)	Duodenum	▪ Gallbladder contraction stimulates pancreatic acinar cell growth ▪ Inhibits gastric emptying	▪ Polypeptides ▪ Amino acids ▪ Fat ▪ Hydrochloride (HCl)	▪ Chymotrypsin ▪ Trypsin
Secretin	Duodenum	▪ Stimulates pancreatic secretion of H_2O and HCO_3 ▪ Bile secretion of HCO_3 ▪ Pepsin secretion ▪ Inhibits gastric acid secretion	▪ Low pH (acid) ▪ Intraluminal duodenal fat	▪ High duodenal pH
Somatostatin	Pancreas	▪ Increases small bowel reabsorption of H_2O and electrolytes ▪ Inhibits cell growth; GI motility; gallbladder contraction; pancreatic, biliary, and enteric secretion of gastric acid; and secretion/action of all GI hormones	▪ Intraluminal fat ▪ Gastric and duodenal mucosa ▪ Catecholamines	▪ Acetylcholine release
Pancreatic polypeptide	Pancreas	▪ Clinical usefulness of pancreatic polypeptide is limited to being a marker for other endocrine tumors of the pancreas	▪ Cephalic—vagus ▪ Gastric—reflexes ▪ Intestinal—food in small bowel	
Neurotensin	Small bowel/colon	▪ Pancreatic secretion ▪ Vasodilation ▪ Inhibits gastric acid secretion	▪ Fat	
Peptide YY	Small bowel/colon	▪ Inhibits gastric acid secretion, pancreatic exocrine secretion, and migrating myoelectric complexes (MMCs)		
Glucagon	Small bowel/colon	▪ Increases glycogenolysis, lipolysis, gluconeogenesis	▪ Low serum glucose	▪ Somatostatin
Motilin		▪ Inhibits MMCs ▪ Increases gastric emptying ▪ Increases pepsin secretion alkaline environment	▪ Vagus ▪ Fat ▪ Intraduodenal	▪ Pancreatic polypeptide

EXAM TIP

Short-chain fatty acids, unlike carbohydrates, are absorbed in the large intestine and can be used as an alternative energy source for persons who suffer from carbohydrate malabsorption.

EXAM TIP

The common causes of small bowel obstruction are: **H**enry **a** te **V**olumes **I**n **C**hicago, **G**aining **S**uccess **N**owhere

Hernia
a dhesions
Volvulus
Intussusception/Ileus
Crohn's disease
Gallstone ileus
SMA syndrome
Neoplasm

Table 9-2. Absorption in the Small Intestine

	Duodenum	Jejunum	Ileum
Water	+	+++	++
Sodium	+	+++	++
Potassium	+	+++	+
Chloride	+	++	+++
Fats	++	+++	+
Proteins	++	+++	+
Carbohydrates	++	+++	+
Bile salts	0	0	+++
Fat-soluble vitamins[a]	+	+++	+
Water-soluble vitamins			
Vitamin B_{12}	0	0	+++
Folic acid	+	+++	++
Ascorbic acid	?	+	+++
Minerals	+++	++	0
Iron	+++	++	0
Calcium	++	+++	+
Magnesium	+	++	++
Zinc[b]	0	+	++

[a] Vitamin K (endogenously produced fraction) absorbed in colon.

[b] Based on animal studies.

Reproduced, with permission, from Wilson JAP, Owyang C. Physiology of digestion and absorption. In Nelson RL, Nyhus LM, eds. Surgery of the Small Intestine. East Norwalk, CT: Appleton & Lange; 1987: 22.

Types

- Partial SBO—only part of the lumen is occluded, allowing some passage of intraluminal contents.
- Complete SBO—no passage of intraluminal contents distally.
- Closed loop obstruction:
  - Blockage of both proximal and distal segments of small intestine.
  - Seen with incarcerated hernia, torsion, adhesions, volvulus.
  - Requires emergent surgery because progression to strangulation is quite rapid.

Risk Factors

- Previous abdominal surgery (most common risk factor).
- Hernia.
- Inflammatory bowel disease (Crohn's disease secondary to stricture formation).
- Diverticular disease.
- Cholelithiasis.
- Ingested foreign body.

Signs and Symptoms

- Colicky abdominal pain.
- Abdominal distention.
- Nausea.
- Vomiting.
- Obstipation.
- Hyperactive bowel sounds.
- Signs of decreased intravascular volume (hemoconcentration, electrolyte abnormalities) secondary to decreased PO intake, vomiting, and accumulation of fluid in bowel lumen and wall (third spacing).

Diagnosis

- Should differentiate between:
  - Mechanical vs. functional
  - Partial vs. complete
  - Simple vs. strangulation
- History should explore possible risk factors that may point to specific etiology.
- Physical exam should include meticulous abdominal exam, search for possible hernias, and examination of stool for gross or occult blood, which may indicate presence of strangulation.
- Confirm by abdominal series:
  - Supine abdominal x-ray: Dilated loops of small intestine with paucity of air in colon.
  - Upright abdominal x-ray: Multiple air-fluid levels in a "stepladder" (Figure 9-1).
  - Upright chest radiograph: Can detect presence of free air under the diaphragm and thus possible bowel perforation.
- Abdominal computed tomography (CT) scan more sensitive and specific than x-rays (Figure 9-2).
- Findings: Transition zone, with dilation of bowel proximally and decompression of the bowel distally, no contrast present distal to transition point, and paucity of gas and fluid in colon.
- Useful in acute setting to rule out other diagnoses as well.

Treatment

If the patient is stable or has partial SBO, give a trial of nonoperative management:

- NPO.
- IV hydration to counter effects of third spacing.
- Nasogastric tube (NGT) for gastric decompression; decreases nausea, vomiting, distention.
- Foley catheter to monitor urine output.
- Monitor electrolytes for signs of hypokalemia, base deficit/metabolic acidosis (signs of ischemia).

- Patients with suspected strangulation need to be resuscitated with fluids prior to surgery.

WARD TIP

As opposed to large bowel obstruction, SBO is rarely caused by neoplasm. If neoplasm is the cause, it is most likely secondary to extrinsic compression as opposed to intraluminal obstruction.

WARD TIP

Features associated with strangulated SBO include:

- Tenderness
- Tachycardia
- Fever
- Markedly elevated WBC count
- Acidosis with elevated lactate level

Beware: These indicators are NOT present in 5–15% of patients with intestinal infarction, especially the elderly and the immunocompromised.

WARD TIP

There are no clinical or laboratory parameters that can reliably differentiate between simple obstruction and strangulated obstruction before the onset of ischemia.

WARD TIP

"Never let the sun rise or set on a small bowel obstruction."
While this adage was strictly adhered to in the past, currently, stable patients, particularly those with partial obstruction, are more likely to be treated nonoperatively. As with any patient, if symptoms worsen, or signs of strangulation develop, the patient should be brought to the OR.



FIGURE 9-1. Upright abdominal x-ray with dilated small-bowel loops and air-fluid levels (arrows) in a stair-step pattern, indicative of small-bowel obstruction. (Reproduced, with permission, from Butler KL, Harisignhani M. Acute Care Surgery: Imaging Essentials for Rapid Diagnosis. New York, NY: McGraw-Hill Education; 2015. Figure 9-2.

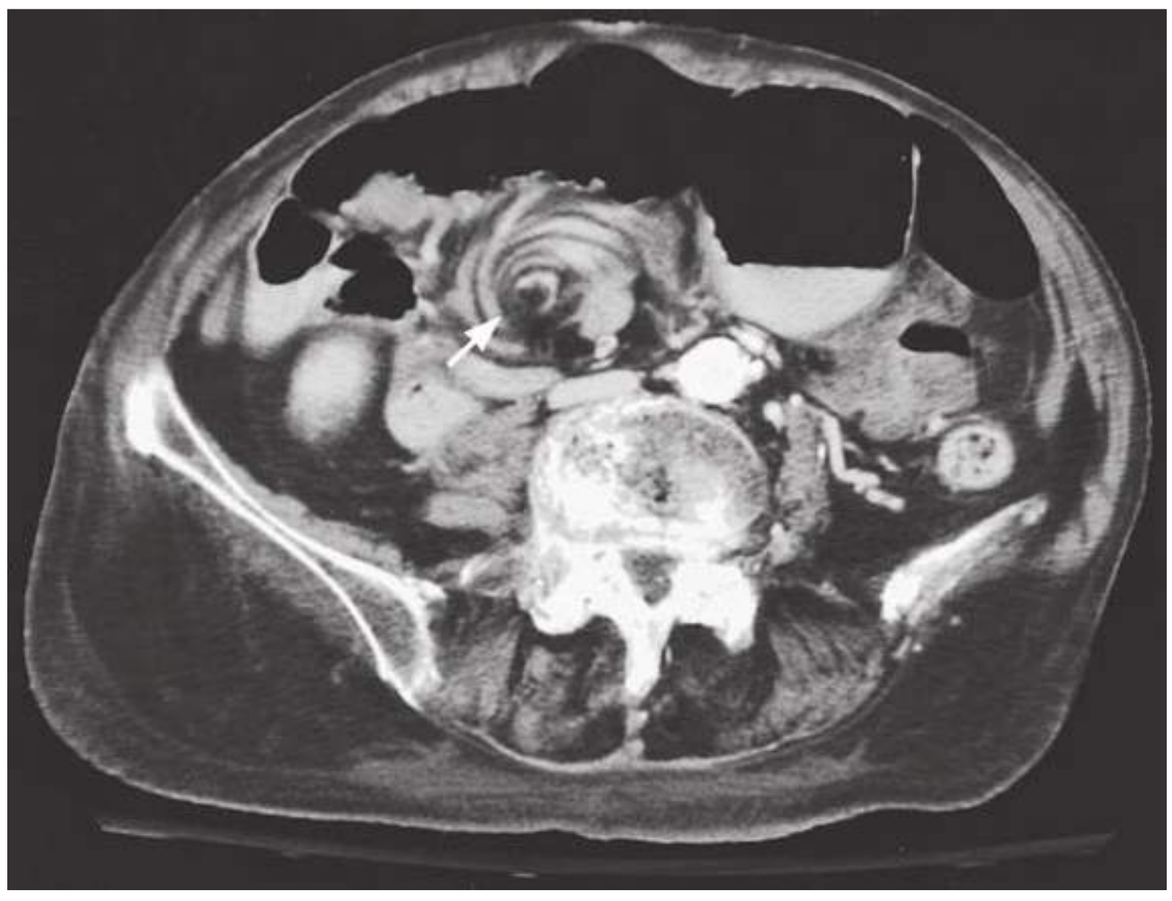


FIGURE 9-2. Contrast abdominal CT scan demonstrating midgut volvulus as a cause of small bowel obstruction.

- If the patient fails conservative management (24 hours without improvement, abdominal tenderness worsens, fever, rising WBC count, other signs of clinical deterioration), then laparotomy should be performed.
- The surgical procedure depends on the cause of the obstruction:
  - Adhesions call for lysis of adhesions (LOA).
  - Hernias should be reduced and repaired or, if contents of sac are strangulated, needs intestinal resection.
  - Cancer requires en bloc resection with lymph node sampling.
  - Crohn's disease requires resection or stricturoplasty of affected area only.

- Whatever the cause, the entire small bowel should be examined, and non-viable intestine should be resected.
- Primary anastomosis should be performed in hemodynamically stable patients who have had small segments of bowel resected.

Crohn's Disease

Definition

Inflammatory bowel disease characterized by transmural granulomatous inflammation involving any part of the GI tract, from mouth to anus, of unknown etiology. The inflammation is discontinuous, resulting in skip lesions, and often leads to fibrosis and ultimately obstruction, as well as to the formation of fistulae.

Epidemiology

- Eighty percent of patients have involvement of small bowel, usually the distal ileum (one third of these patients just have ileitis).
- Fifty percent of patients have involvement of both the ileum and colon.
- Twenty percent of patients have involvement of the colon only. Differentiate from ulcerative colitis because Crohn's disease patients tend to have rectal sparing.
- One third of patients have perianal disease as well.
- Diagnosis most common between ages 15 and 40, although there is a second peak between 50 and 80 years of age (bimodal distribution).

Risk Factors

- Jewish descent.
- Positive family history.
- Urban dwelling.
- Smoking.

Signs and Symptoms

Typical symptoms include:

- Crampy abdominal pain (typically right lower quadrant [RLQ]), diarrhea, weight loss most common symptoms.
- Fever, fatigue.
- Bleeding (hemoccult + stools common, but gross lower GI bleeding less common than in ulcerative colitis).
- Perianal disease including skin tags, anal fissures, perirectal abscesses, and anorectal fistulae.
- Signs and symptoms of intestinal perforation and/or fistula formation (e.g., combination of localized peritonitis, fever, abdominal pain, tenderness, and palpable mass on physical exam).
- Extraintestinal manifestations include oral involvement (e.g., aphthous ulcers), joint involvement (e.g., arthritis), ocular involvement, dermatologic involvement (e.g., erythema nodosum, pyoderma gangrenosum), hepatobiliary involvement (e.g., sclerosing cholangitis).

Diagnosis

- Typical history of prolonged diarrhea with abdominal pain, weight loss, and fever with or without gross bleeding.
- Physical exam can be nonspecific or suggestive of Crohn's disease, such as perianal skin tags, sinus tracts, or a palpable abdominal mass.

EXAM TIP

Typical scenario: A 5-year-old child presents with increasing irritability, colicky abdominal pain, and rectal bleeding with stools that have a currant jelly appearance. A tubular mass is palpated in the right lower quadrant. Upright abdominal x-ray shows air-fluid levels with a stepladder pattern. Think: Intussusception. Barium enema is both diagnostic and therapeutic.

WARD TIP

Differential diagnosis of RLQ pain:

- Appendicitis.
- Crohn's disease.
- Infectious colitis—Yersinia, Campylobacter.
- Gynecologic pathology (ovarian torsion, abscess, ectopic pregnancy).
- Ischemic colitis.

- Colonoscopy (with biopsy), visualization of the terminal ileum may reveal focal ulcerations adjacent to areas of normal mucosa along with a cobblestone appearance to the intestinal mucosa.
- Esophagogastroduodenoscopy (EGD) for proximal disease.
- Imaging (small bowel contrast studies) is helpful in characterizing length of involvement and areas of stricture, especially in parts of small bowel that are inaccessible via colonoscopy.
- Radiographic appearance: Mucosal nodularity, narrowed lumen, ulceration, string sign, presence of abscesses and fistulae.

WARD TIP

Unlike ulcerative colitis, which can be cured with surgical resection, Crohn's disease is incurable surgically.

Treatment

- Medical: Corticosteroids, aminosalicylates (sulfasalazine, 5-ASA), immune modulators (azathioprine, 6-mercaptopurine, cyclosporine), infliximab (anti-TNF-α), metronidazole.
- Many patients require surgery to relieve symptoms that do not respond to drugs, or to treat complications such as obstruction, abscesses, fistulae, perforation, perianal disease, or cancer.
- Surgery should be avoided if possible, since Crohn's disease is not curable by surgery as opposed to ulcerative colitis.
- Surgical resection of an affected area does not preclude future disease development in adjacent or distant parts of the bowel.
- Surgical procedure depends on indication:
  - One third of patients require surgery to relieve intestinal obstruction by strictures, either via segmental small bowel resection or stricturoplasty (Figures 9-3 to 9-5).
  - For fistulae: Seton placement and drainage, fistulotomy, fistulectomy.
  - For extensive colonic disease: Total colectomy with end ileostomy.
  - For cancer: En bloc resection with lymph node dissection.

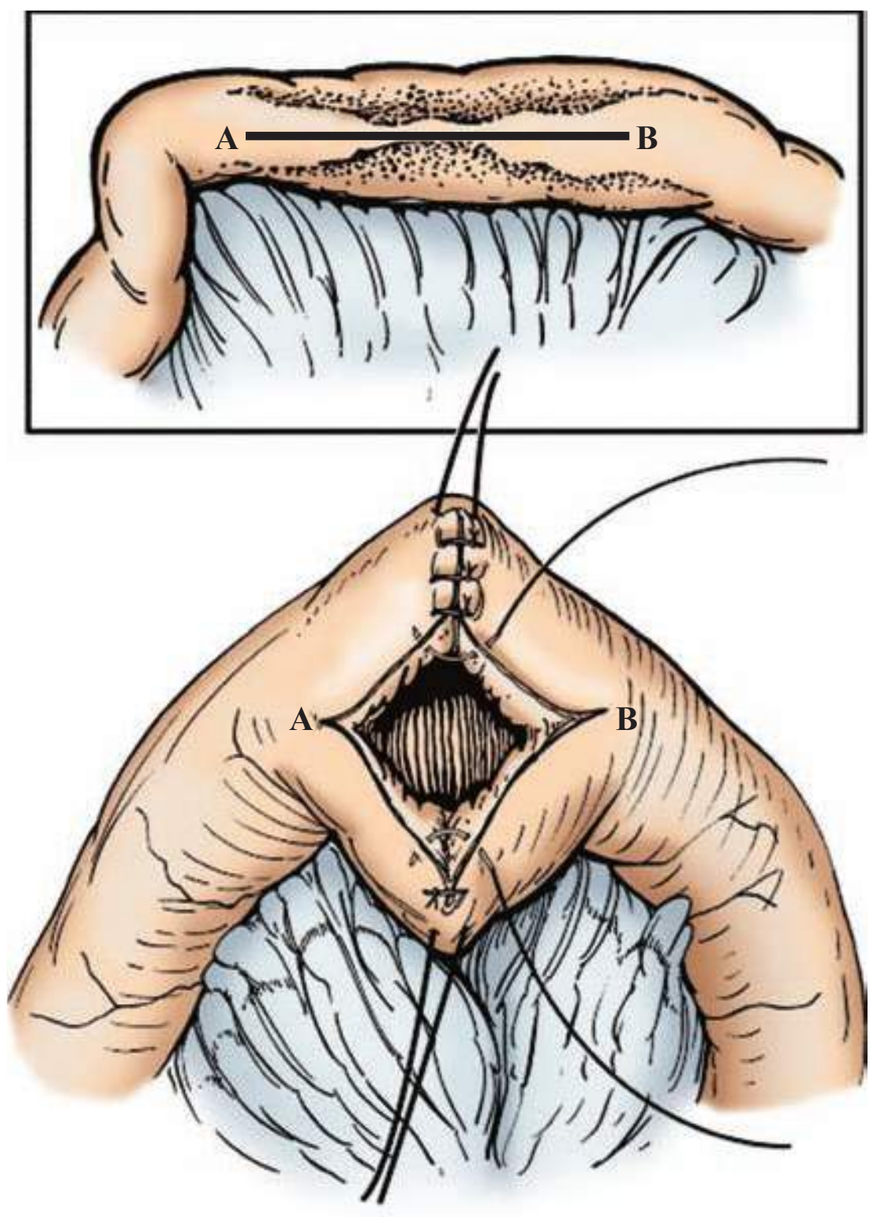


FIGURE 9-3. Heineke–mikulicz strictureplasty. (Reprinted, with permission, from Michelassi F, Hurst RD, Fichera A. Crohn's disease. In Zinner MJ, Ashley SW, eds. Maingot's Abdominal Operations. 11th ed. New York: McGraw-Hill; 2007: 533.)

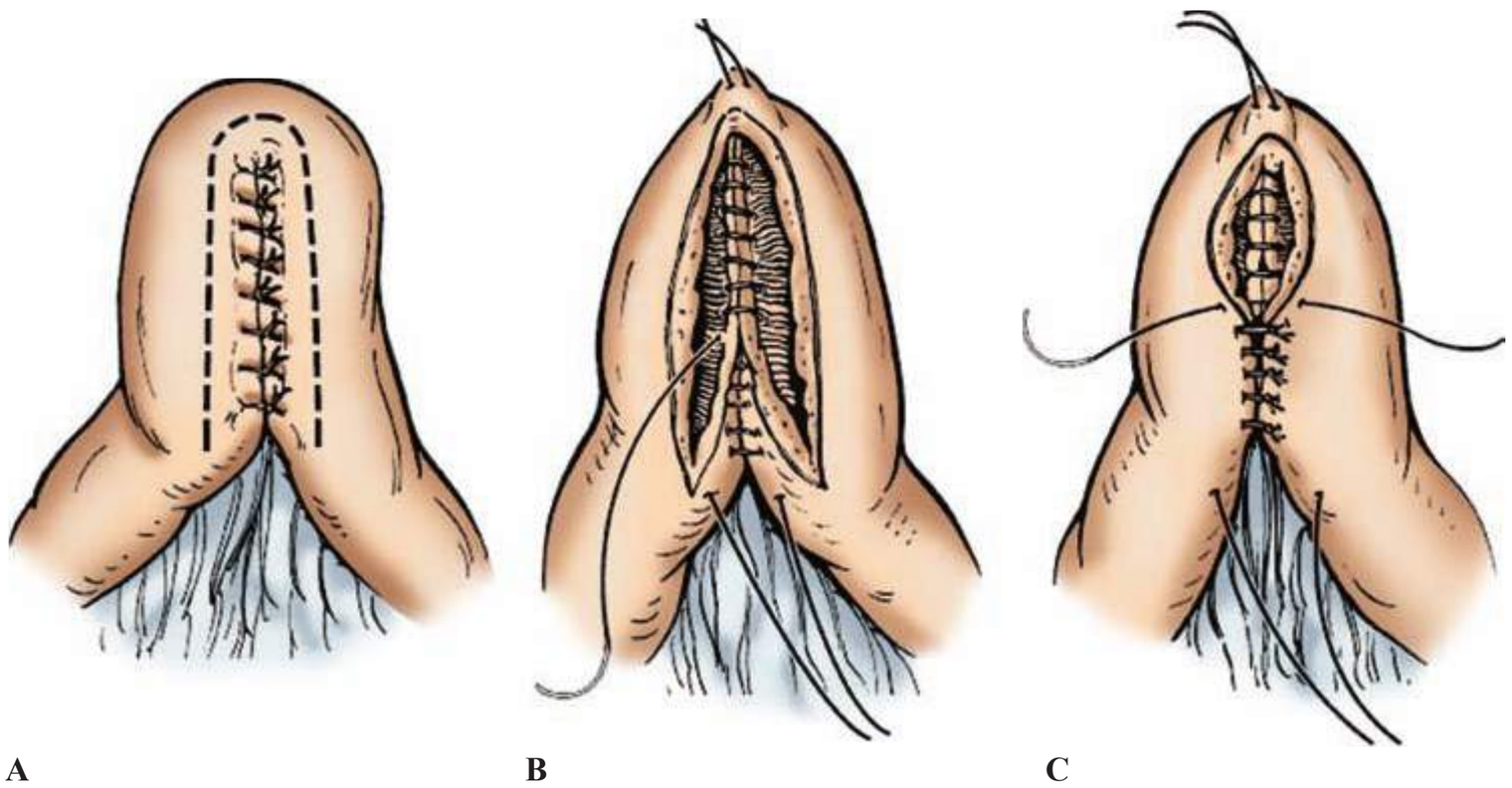


FIGURE 9-4. Finney strictureplasty. (Reprinted, with permission, from Michelassi F, Hurst RD, Fichera A. Crohn's disease. In Zinner MJ, Ashley SW, eds. Maingot's Abdominal Operations. 11th ed. New York: McGraw-Hill; 2007: 534.)

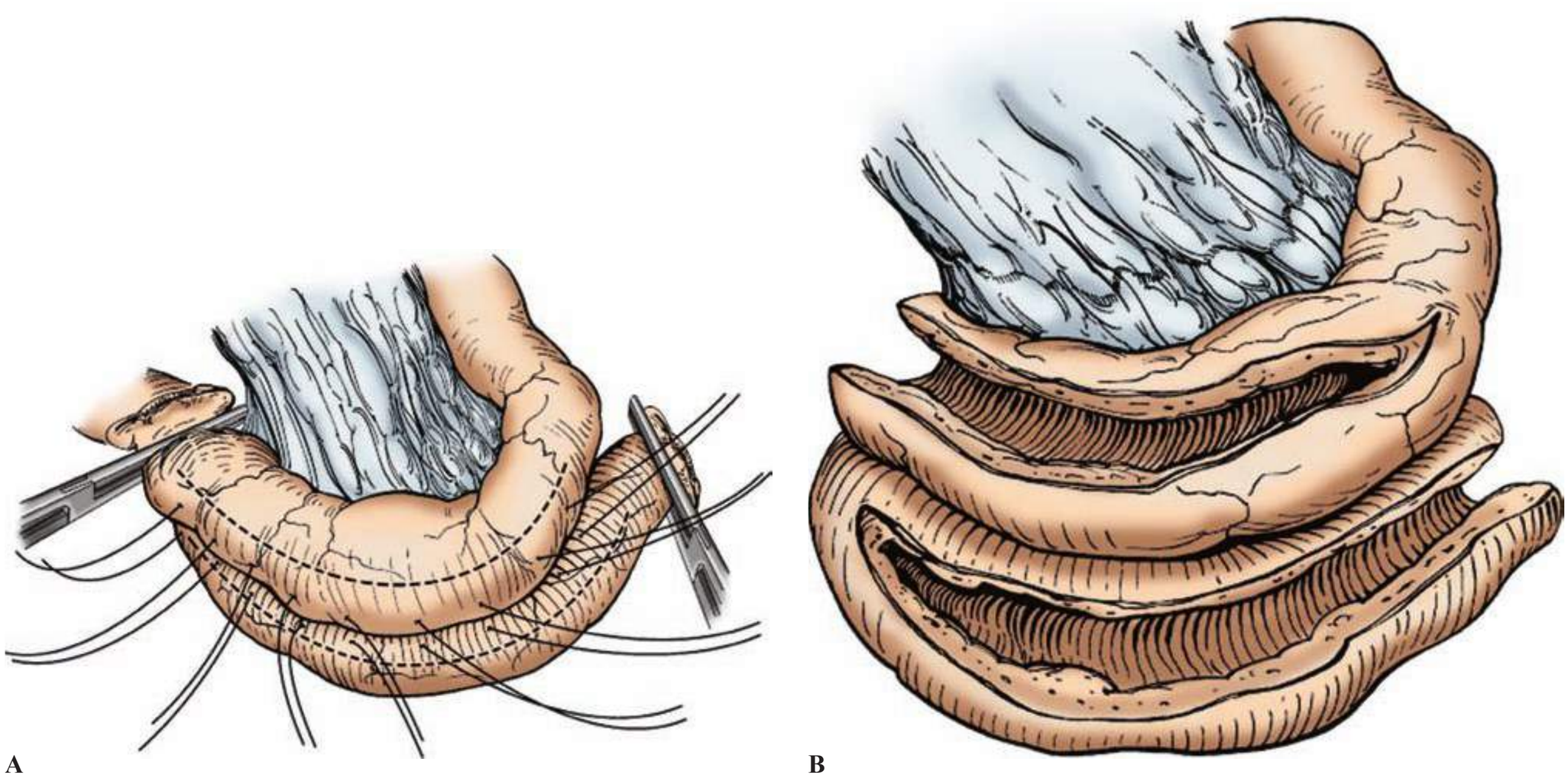


FIGURE 9-5a AND B. Isoperistaltic side-to-side strictureplasty. (Reproduced, with permission, from Zinner MJ, Ashley SW. Maingot's Abdominal Operations. 12th ed. New York, NY: McGraw-Hill Education; 2013. Figures 33-7 and 33-8.

Prognosis

- Typical course is one of intermittent exacerbations followed by periods of remission.
- Ten to 20% of patients experience prolonged remission after initial presentation.
- Approximately 80% of patients ultimately require surgical intervention.
- Like ulcerative colitis, there is an increased risk of colon cancer in patients with long-standing Crohn's colitis.
- Resection with anastomosis has 10–15% clinical recurrence rate per year.
- Total colectomy with ileostomy has 10% recurrence rate over 10 years in remaining small bowel.

Benign neoplasms of Small intestine

Incidence

- Adenomas > leiomyomas > lipomas (but leiomyomas most likely to cause symptoms).
- Most small intestinal benign neoplasms are found in the duodenum.
- Most common cause of adult intussusception.
- Most patients in 5th or 6th decade of life.

Risk Factors

- Hereditary syndromes:
  - Peutz–Jeghers syndrome (hamartomatous polyps).
  - Gardner syndrome (adenoma).
  - Familial adenomatous polyposis (adenoma).
- Consumption of red meat and salt-cured foods.

WARD TIP

Intermittent obstruction is the most common presentation in a symptomatic patient with a small bowel neoplasm. It can be caused by either narrowing of the small bowel lumen or secondary to intussusception, with the neoplasm serving as the lead point.

Signs and Symptoms

- Most are asymptomatic until they become large.
- Intermittent obstruction: Crampy abdominal pain, distention, nausea, and vomiting.
- Occult or overt bleeding.
- Palpable abdominal mass.
- Obstructive jaundice (periampullary lesion).

WARD TIP

enteroclysis is a double-contrast study that involves passing a tube into the proximal small intestine and injecting barium and methylcellulose. It can detect tumors missed on conventional small bowel follow-through. **extended small bowel enteroscopy** (Sonde enteroscopy) is much like push enteroscopy, but involves advancement of the enteroscope by peristalsis. It visualizes up to 70% of the small bowel mucosa and detects tumors missed by enteroclysis.

Diagnosis

- Endoscopy:
  - EGD: Can visualize proximal duodenum; most duodenal neoplasms are found incidentally on EGD.
  - Endoscopic ultrasound (EUS): Can offer more information such as the depth of intestinal wall involved.
- Radiographic:
  - Small bowel series—low sensitivity.
  - CT scan.
  - Enteroclysis: Test of choice; high sensitivity; used to detect tumors in distal small intestine.
- Majority of patients: Small bowel series with follow-through followed by enteroclysis.
- High-risk patients: Enteroclysis.

Types

See Table 9-3.

Treatment

- All symptomatic lesions should be resected, either surgically or endoscopically.
- Tumors located in proximal duodenum, even asymptomatic lesions, should be removed either endoscopically (< 1 cm) or surgically (> 2 cm).
- Tumors in second portion of duodenum, near ampulla, may require pancreaticoduodenectomy (Whipple procedure).

malignant neoplasms of Small intestine

- Rare.
- Adenocarcinoma > carcinoid > gastrointestinal stromal tumor (GIST) > lymphoma.

Table 9-3. Benign Neoplasms of the Small Intestine

type	risk factors	Signs and Symptoms	location	treatment
Adenoma (35% of benign small bowel tumors)	Gardner's syndrome (GS)	Obstruction	Duodenum (20%)	Endoscopic or surgical excision For GS and familial adenomaous polyposis (FAP): ▪ Screening EGD 2nd–3rd decade ▪ Adenomas resected endoscopically ▪ Adenoma recurrence requires pancreaticoduodenectomy (risk of ampullary carcinoma)
		Bleeding	Jejunum (30%)	Excision
	FAP		Ileum (50%)	Excision
Leiomyoma		Obstruction Bleeding	Jejunum	Excision
Lipoma		Obstruction Incidental finding	Duodenum Ileum	Excision is required only if symptomatic
Hamartoma	Peutz–Jeghers syndrome	▪ Recurrent colicky abdominal pain (from intermittent intussusception) ▪ Obstruction ▪ Bleeding		▪ Resection of segment responsible for symptoms
Hemangioma (3–4% of benign small bowel tumors)		Bleeding		
Fibroma		Obstruction Asymptomatic mass		

- Risk factors, signs and symptoms, and diagnosis essentially the same as benign neoplasms of small intestine (see Table 9-4):
  - Adenocarcinoma: Crohn's disease, celiac disease, familial adenomatous polyposis (FAP), Peutz–Jeghers syndrome.
  - Lymphoma: Celiac disease, immunodeficiency states, autoimmune disorders.

Treatment

- Wide en bloc resection of involved intestine.
- For adenocarcinomas, wide local excision of the intestine with its accompanying mesentery is performed along with regional lymph nodes.
- GISTs usually treated with segmental resection of affected intestine and Gleevec (imatinib), a tyrosine kinase inhibitor.
- Patients with duodenal lesions may require a Whipple procedure.
- Bypass may be required for palliation.

EXAM TIP

Typical scenario: A patient presents with pigmented spots on his lips and a history of recurrent colicky abdominal pain. What is the cause of his abdominal pain? Think: Peutz–Jeghers syndrome. The hamartomatous polyps are likely causing intermittent intussusception.

Table 9-4. Malignant Neoplasms of the Small Intestine

Type	Risk Factors	Signs and Symptoms	Location (in Small Gut)
Adenocarcinoma (25–50% of primary small bowel malignancies)	Crohn's disease, celiac disease, FAP, Peutz-Jeghers syndrome	Obstruction	Duodenum (most found in duodenum)
		Bleeding	Ileum (in Crohn's disease most adenocarcinomas found in ileum)
		Mass	
Carcinoid (up to 40% of primary small bowel malignancies)		Often asymptomatic	Ileum
		Obstruction Carcinoid syndrome	
GIST (CD 117 [c-kit])		Hemorrhage	No regional preference
Lymphoma	Celiac disease	Obstruction Perforation	Ileum
	Immunosuppression	Weight loss	
	Autoimmune disease	Pain Fatigue Mass Bleeding	
Sarcoma (most common leiomyosarcoma)		Pain	Jejunum
		Bleeding	Ileum
		Obstruction (late symptom)	Meckel's diverticulum
Neuroendocrine		Mass	Proximal small intestine
		Hormone-specific symptoms	
Metastatic	History of melanoma, breast, lung, ovarian, colon, or cervical cancer	Obstruction; bleeding	

- Localized lymphoma is treated with segmental resection of the intestine, and neighboring mesentery.
- Diffuse lymphoma is the only situation where chemotherapy, rather than surgical resection, should be the primary therapy.

WARD TIP

Small bowel is frequently affected by metastasis or invasion from cancers originating in other organs, particularly melanoma.

Prognosis

- Overall 5-year survival: ≤ 20%.
- Five-year survival by tumor type:
  - Complete resection of duodenal adenocarcinoma: 50–60%.
  - Complete resection of jejunal/ileal adenocarcinoma: 5–30%.

- GIST: 30–40%.
- Localized lymphoma: 60%.

carcinoid

Definition

- Malignant tumor of enterochromaffin cell origin, part of amine precursor uptake and decarboxylation (APUD) system.
- Most common in appendix.

Incidence

- Carcinoid tumors represent between 29 and 40% of primary small bowel malignancies.
- Peak incidence between 50 and 70 years of age.
- More than 90% diagnosed in GI system:
- The appendix is the most common site of GI carcinoid followed by small bowel followed by the rectum.

Signs and Symptoms

- Slow growing; therefore, frequently asymptomatic, and are usually found incidentally.
- In symptomatic patients, vague abdominal pain is the most common symptom.
- Intermittent obstruction—25% of patients.
- Rectal bleeding (from rectal carcinoid), pain, weight loss.
- Carcinoid syndrome: Approximately 10% of cases:
  - Due to production of serotonin, bradykinin, or tryptophan by tumor and exposure of these products to systemic circulation prior to breakdown by the liver.
  - Characterized by cutaneous flushing, sweating, watery diarrhea, wheezing, dyspnea, valvular lesions (right > left).

Diagnosis

- Most found incidentally during radiographic studies, appendectomy, or surgery for intestinal obstruction.
- If patient has carcinoid syndrome:
  - 5-HIAA (hydroxyindolacetic acid) in 24-hour urine collection.
  - Plasma chromogranin A—independent predictor of prognosis.
  - Pentagastrin provocation test: Pentagastrin administration induces flushing (used in patients with only marginally elevated 5-HIAA, but who describe flushing symptoms).
  - Otherwise diagnosed as any other small bowel neoplasm.

Treatment

- Medical: Serotonin antagonists (e.g., cyproheptadine) or somatostatin analogues (e.g., octreotide) for symptoms of carcinoid syndrome.
- Surgical:
  - Appendiceal carcinoid < 2 cm: Appendectomy.
  - Appendiceal carcinoid > 2 cm: Right hemicolectomy.
  - Small intestinal carcinoid: Resect tumor with mesenteric lymph nodes, as well as inspection of entire small bowel for synchronous lesions.
  - Otherwise, resect tumor and any solitary liver metastasis considered resectable.

EXAM TIP

The carcinoid syndrome develops when the tumor produces amines and peptides outside of the portovenous circulation. Classically, appendiceal and small intestinal carcinoids cause the carcinoid syndrome only after they have metastasized to the liver.

WARD TIP

Patients who present with carcinoid syndrome are typically not surgical candidates since they already have extensive metastatic disease.

EXAM TIP

Typical scenario: A 60-year-old male presents with a history of cutaneous flushing, diarrhea, wheezing, and an unintentional 15-lb weight loss. Think: Carcinoid syndrome; the wheezing is a clue that the lesion may be endobronchial. Order a 24-hour urine 5-HIAA level to confirm the diagnosis.

Prognosis

- Overall survival roughly 54%.
- Five-year survival after palliative resection is 25%; after curative resection is 70%.

Fistula

Definition

A communication between two epithelialized surfaces. It can form between two parts of the GI or genitourinary (GU) tract, an internal fistula (e.g., choledochoduodenal or colovesical), or between an internal organ and an outer epithelialized surface (e.g., enterocutaneous), an external fistula.

Risk Factors

- Previous abdominal surgery (most common—80%).
- Diverticular disease.
- Crohn's disease.
- Colorectal cancer.

Signs and Symptoms

- Iatrogenic fistulae usually appear 5–10 days postoperatively.
- If associated with an abscess, can be accompanied by fever and leukocytosis.
- Drainage of succus entericus (bowel contents) from skin (enterocutaneous fistula).
- Diarrhea, usually secondary to malabsorption (enteroenteric; especially if between proximal and distal small bowel or colon since a large portion of the small bowel absorptive surface is bypassed).
- Pneumaturia and symptoms of urinary tract infection (colovesicular fistula).

WARD TIP

Proximal fistulas cause more problems than distal fistulas because the draining contents are more acidic, more fluids and electrolytes are lost, and more absorptive area is lost.

Diagnosis

- CT with enteral contrast shows leakage of contrast from intestinal lumen.
- Small bowel series.
- Enteroclysis.
- Fistulogram: Contrast is injected directly into fistula tract.

Treatment

- Stabilization: Manage electrolyte abnormalities, fluid losses, and nutritional status.
- Drainage of abscesses (if present).
- Allow time for spontaneous closure:
  - Bowel rest.
  - Provide nutrition (usually total parental nutrition [TPN]).
  - Consider octreotide (somatostatin analogue) for high-output and pancreatic fistulas.
- If 6–8 weeks pass without improvement, then surgery should be performed to resect the fistula tract, together with the segment of small bowel from which it originates.

WARD TIP

High output fistulas drain > 500 mL/24 hrs and are proximal (stomach, duodenum, proximal small bowel). Low output fistulas drain < 500 mL/24 hrs and are distal (distal small bowel and colon).

Prognosis

- Enterocutaneous fistulas have 15–20% mortality related to complications of sepsis and underlying disease.
- Surgery is associated with considerable morbidity and a high recurrence rate.

mesenteric ischemia

Definition

- Reduction in blood flow to the small bowel secondary to a variety of mechanisms.
- Can result in two distinct conditions: acute mesenteric ischemia and chronic mesenteric ischemia.

acute mesenteric ischemia (ami)

- Rapid onset of intestinal hypoperfusion.
- Can be caused by arterial occlusion (usually single artery):
  - SMA embolism—50% of all AMI.
  - SMA thrombosis (acute)—15–25% of all AMI.
  - Vasospasm (nonocclusive mesenteric ischemia)—20–30% of all AMI.
  - Venous obstruction (either thrombosis or strangulation usually of SMV)—5% of all AMI.
- Regardless of cause, AMI can lead to mucosal sloughing within 3 hours of onset, and to intestinal infarction 6 hours after onset.

Risk Factors

- Cardiac: Low cardiac output states, cardiac arrhythmias, severe cardiac valvular disease, recent myocardial infarction.
- Age.
- Atherosclerosis.
- Hypercoagulable states.

Signs and Symptoms

- Abdominal pain out of proportion to tenderness on physical exam (hallmark).
- Pain is colicky and diffuse, typically in mid-abdomen.
- Can be associated with nausea, vomiting, and diarrhea.
- Following infarction, peritonitis (rebound and rigidity), abdominal distention, and passage of bloody stools occur.

Diagnosis

- High clinical suspicion especially if history of cardiac disease or hypercoagulability.
- No lab test is sensitive for diagnosis of AMI. Findings may include leukocytosis, acidosis, hemoconcentration, and occult blood in stool.
- If there are peritoneal signs, then patient should undergo emergent laparotomy.
- Otherwise, pursue diagnostic tests:
  - Mesentric angiography: Most sensitive and specific method for diagnosing AMI, albeit invasive.
  - CT scan with IV contrast:
    - Good initial test for most patients with possible AMI.
    - Can rule out other causes of abdominal pain.
    - Can reveal evidence of ischemia of intestine and mesentery.
    - Can reveal occlusion/stenosis of vasculature.

Therapy

- Stabilization of patient:
  - Hemodynamic monitoring and fluid resuscitation.
  - Correction of electrolyte abnormalities, acidosis.

WARD TIP

Factors that can keep a fistula tract open: FRIeNDS
Foreign body
Radiation
Inflammation/Infection
epithelialization
Neoplasm
Distal obstruction
Short segment fistula (< 2.5-cm fistula tract)

EXAM TIP

SMA is the most common vessel involved in AMI. Most emboli that cause AMI originate in the heart (left atrial or left ventricular thrombi). Ninety-five percent of patients with AMI will have a history of cardiovascular disease (atrial fibrillation or myocardial infarction).

WARD TIP

- CT has somewhat of a low sensitivity for detecting **arterial** AMI. Therefore, if a patient has high suspicion for AMI, then patient should undergo angiography despite negative CT results.
- CT has high sensitivity (> 90%) for detecting acute mesenteric **venous thrombosis.**

- Broad-spectrum antibiotics.
- Placement of a nasogastric tube for gastric decompression.

- Patient with peritoneal features: Midline laparotomy with assessment of intestinal viability.
  - For embolic causes: Intraoperative embolectomy.
  - For CA/SMA thrombosis: Bypass of site of obstruction using a saphenous vein graft (from supraceliac aorta to distal SMA).
- Therapy via angiography includes intra-arterial vasodilators (papaverine) or thrombolytic agents (streptokinase, urokinase, tPA), angioplasty, placement of a stent, and embolectomy.
- Venous thrombosis requires immediate anticoagulation (heparin).



WARD TIP

Thrombolytic therapy for intra-arterial emboli is most successful when initiated within 8–12 hours of symptom onset.

Prognosis

- Acute arterial mesenteric ischemia mortality rate: 59–93%.
- Acute mesenteric venous thrombosis mortality rate: 20–50% (30% recurrence rate if not anticoagulated).



WARD TIP

Patients with acute mesenteric venous thrombosis should be evaluated for hereditary and acquired thrombophilias.

chronic mesenteric ischemia (cmi)

- Insidious, episodic, or constant state of intestinal hypoperfusion.
- Rarely leads to infarction of small bowel due to development of collateral circulation over time.
- Can be caused by:
  - Arterial ischemia (most common): Associated with atherosclerosis of more than one mesenteric and splanchnic vessels.
  - Venous thrombosis: Thrombosis of portal or splenic veins, leading to portal hypertension with subsequent development of esophageal varices and splenomegaly.
  - Vasculitis.

Risk Factors

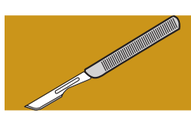
- Atherosclerotic vascular disease (half of patients have history of peripheral vascular disease or coronary artery disease).
- Smoking.

Signs and Symptoms

- "Intestinal angina": Dull, crampy, postprandial abdominal pain leading to food aversion and weight loss (hallmark).
- Patients with chronic venous thrombosis may be asymptomatic due to collateral formation, or may present with esophageal variceal bleeding.

Diagnosis

- High clinical suspicion.
- Must rule out other causes of chronic abdominal pain, weight loss, and food aversion, particularly malignancy.
- Diagnosis is supported by demonstration of high-grade stenoses in multiple mesenteric vessels.
- Diagnostic tests:
  - Angiography: Gold standard.
  - CT and magnetic resonance (MR) angiography: Good initial tests since they can identify whether a stenosis is present and serve as a guide for angiography.
  - Mesenteric duplex ultrasonography: Can be used as a screening test to detect stenoses of the celiac and superior mesenteric arteries.



OR TIP

CMI can occur due to compression of celiac artery by diaphragm—celiac artery compression syndrome or median arcuate ligament syndrome. Treatment is release of the arcuate ligament and bypass of persistent stricture.

Therapy

- Arterial CMI:
  - Surgical revascularization:
    - Aortomesenteric bypass graft
    - Mesenteric endarterectomy
  - Percutaneous transluminal angioplasty (PTA) with or without placement of a stent:
    - Less relief of symptoms and less durability than surgery.
    - Serves as alternative to surgery for those patients who are not optimal surgical candidates due to considerable comorbidities.
- Chronic venous mesenteric thrombosis: Anticoagulation (heparin).

Prognosis

- Perioperative mortality rates range from 0 to 16%.
- Initial relief of symptoms with surgery: 90%.
- Recurrence rate:
  - Following surgery: < 10%.
  - Following PTA: 10–67%.

Short Bowel Syndrome

Definition

- Presence of < 200 cm of small bowel in adult patients.
- Results in decreased small bowel absorption, leading to diarrhea, malnutrition, and dehydration.

etiologies

- Adults: Acute mesenteric ischemia, Crohn's disease, malignancy.
- Pediatrics: Volvulus, intestinal atresia, necrotizing enterocolitis.

Pathophysiology

- Malabsorption occurs following resection of 50–80% of small bowel.
- Presence of intact colon, as well as an ileocecal valve, also decreases severity of malabsorption.
- Resection of ileum is associated with increased malabsorption secondary to inability to absorb bile salts and vitamin B_{12}.
- Small bowel adaptation period lasts approximately 1–2 years following surgery.

Therapy

- Medical:
  - Repletion of fluid and electrolytes: Initially, most patients require TPN.
  - Proton pump inhibitors/H_2 blockers to decrease gastric acid secretion.
  - Antimotility agents to decrease transit through small bowel.
  - Octreotide may be used to decrease intestinal secretions.
- Surgical:
  - Restoration of intestinal continuity in patients with stomas, in order to increase absorptive capacity.
- Surgeries aimed at slowing transit through the small intestine include:
  - Segmental reversal of the small bowel.
  - Placement of a segment of colon between two segments of small bowel.
  - Creation of artif cial small bowel valves.

EXAM TIP

Typical scenario: A 70-year-old male with a history of peripheral vascular disease and hyperlipidemia presents to the emergency department with severe, diffuse abdominal pain. His blood pressure is 170/100 and his pulse is 90 bpm. Supine abdominal radiograph shows free air in the abdomen and within the wall of the small intestine. What is the most likely diagnosis? Think: Small bowel infarction.

WARD TIP

TPN is associated with considerable morbidity including catheter sepsis, liver and kidney failure, and venous thrombosis.

- Surgeries aimed at lengthening the small bowel include:
  - Longitudinal intestinal lengthening and tailoring (LILT).
  - Serial transverse enteroplasty procedure (STEP).
  - Small bowel transplantation:
    - One hundred performed in the United States each year.
    - Indicated for patients with life-threatening intestinal failure, or complications from TPN.
    - Eighty percent of survivors have full intestinal function without need for TPN.
    - Signif cant risks including acute or chronic rejection, as well as CMV infection.

Prognosis

- Fifty to 70% of patients who initially require TPN can eventually be completely weaned off of TPN.
- Pediatric patients are more likely than adults to gain independence from TPN.

HIGH-YIELD FACTS IN

Colon, Rectum, and anal Canal

EMBRYOLOGY AND ANATOMY 144
Embryology 144
gross Anatomy 144
Colon 144
rectum 144
Anus 145
blood Supply 145
lymphatic drainage 146
innervation 146
histology 146
microbiology 146
Physiology 146
general 146
motility 146
defecation 147
Disorders of Motility 147
irritable bowel syndrome (ibs) 147
Constipation 147
diarrhea 148
Colitis 148
pseudomembranous Colitis 148
radiation-induced Colitis 148
ischemic Colitis 149
inflammatory bowel disease: ulcerative Colitis 150
Diverticular Disease 151
definition 151
incidence 151
risk factors 152
Signs And Symptoms 152
diagnosis 152
treatment 153
prognosis 153
Lower GI Bleed (LGIB) 153
definition 153
management 153
Large Bowel Obstruction 154
incidence 154
Signs And Symptoms 154
diagnosis 154
treatment 155
Volvulus 155
definition 155
incidence 155
risk factors 155
Signs And Symptoms 155
diagnosis 155
treatment 155
Pseudo-obstruction (Ogilvie Syndrome) 156
definition 156
incidence 156
risk factors 156
Signs And Symptoms 156
diagnosis 156
treatment 156
Benign Tumors of the Large Bowel 157
Colorectal polyps 157
polyposis Syndromes 157
Colorectal Carcinoma (CRC) 158
incidence 158
risk factors 158
Adenoma-Carcinoma Sequence 159
Perianal and Anal Problems 161
hemorrhoids 161
Anal fissure 163
Anorectal Abscess 163
Anorectal fistulas 164
pilonidal disease 165
Anal Cancer 165
definition 165
incidence 165
risk factors 165
Signs And Symptoms 165
diagnosis 165
treatment 166
prognosis 166

EMBRYOLOGY AND ANATOMY

Embryology

- Origin: Embryonic midgut (up to mid-transverse colon) and hindgut (rest of colon, and proximal anus). Distal anus derived from ectoderm.
- The dentate line marks the transition between hindgut and ectoderm.

WARD TIP

In development, the midgut loop rotates 270° counterclockwise around the axis of the superior mesenteric artery (SMA). Developmental anomalies include malrotation or failure of right colon to elongate.

Gross Anatomy

See Figure 10-1.

Colon

- Extends from the ileocecal valve to the rectum; consists of: ascending colon, transverse colon, descending colon, sigmoid colon.
- 3–5 feet in length.
- Cecum is widest; the colon progressively narrows distally.
- Unlike the small intestine, the colon has taenia coli, haustra, and appendices epiploicae (fat appendages that hang off antimesenteric side of colon).
  - Taenia coli—three distinct bands of longitudinal muscle—converge at the appendix and spread out to form a longitudinal muscle layer at the proximal rectum.
  - Retroperitoneal attachments of ascending and descending colon fix it to the posterior abdominal wall.
- Retroperitoneal: Ascending colon, descending colon, posterior hepatic and splenic flexures.
- Intraperitoneal: Cecum, transverse colon, sigmoid colon.
- One end of omentum attaches to anterior-superior aspect of transverse colon; other end attaches to stomach.

EXAM TIP

The blood supply is based on embryology:

- Midgut: SMA
- Hindgut: Inferior mesenteric artery (IMA)
- Distal anus: Internal pudendal artery branches

Rectum

- 12–15 cm in length.
- Rectum has distinct peritoneal covering.

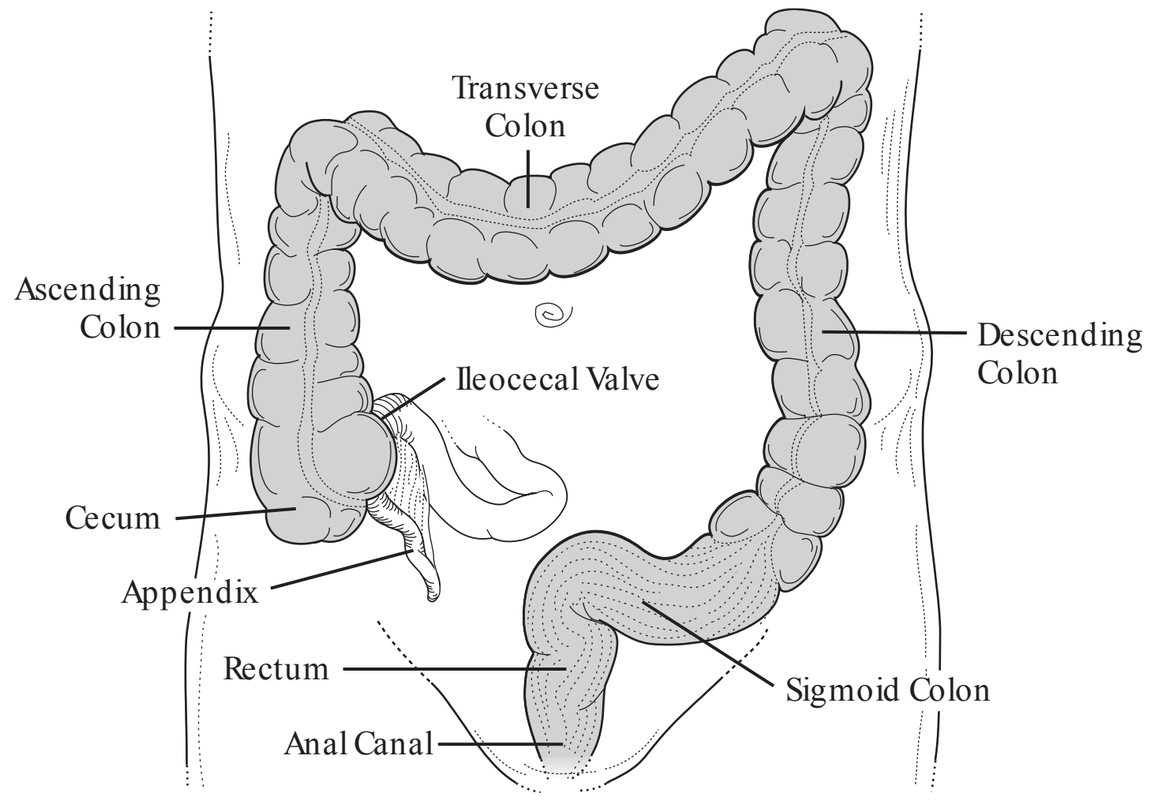


FIGURE 10-1. Bowel anatomy.

- Fascia:
  - Waldeyer's fascia: Rectosacral fascia that extends from S4 vertebral body to rectum.
  - Denonvilliers' fascia: Anterior to lower third of rectum.
- Pelvic floor: Levator ani (composed of pubococcygeus, iliococcygeus, and puborectalis muscles); innervated by S4 nerve.

Anus

- Anal canal runs from pelvic diaphragm to anal verge (junction of anoderm and perianal skin).
- Dentate line: A mucocutaneous line that separates proximal, pleated mucosa from distal, smooth anoderm (1–1.5 cm above anal verge).
- Anal mucosa proximal to dentate line lined by columnar epithelium; mucosa distal to dentate line (anoderm) lined by squamous epithelium and lacks glands and hair.
- Columns of Morgagni: 12–14 columns of pleated mucosa superior to the dentate line separated by crypts. Perianal glands discharge their secretions at the base of the columns.
- Anal sphincter:
  - Internal: Consists of specialized rectal smooth muscle (from inner circular layer); involuntary, contracted at rest, responsible for 80% of resting pressure.
  - External: Consists of three loops of voluntary striated muscle; a continuation of puborectalis muscle; responsible for 20% of resting pressure and 100% of voluntary pressure.

blood supply

arterial

- **Superior mesenteric artery (SMA):** Supplies the cecum, ascending colon, and proximal two thirds of the transverse colon via the ileocolic, right colic, and middle colic arteries, respectively.
- **Inferior mesenteric artery (IMA):** Supplies the distal two thirds of the transverse colon, sigmoid colon, and superior rectum via the left colic, sigmoidal, and superior rectal (hemorrhoidal) arteries, respectively.
- **Internal iliac artery:** Supplies the middle and distal rectum via the middle rectal and inferior rectal arteries, respectively (the inferior rectal artery is a branch of the internal pudendal artery).
- **Internal pudendal artery:** Supplies the anus; is a branch of the internal iliac artery.

Venous

- **Superior mesenteric vein (SMV):** Drains the cecum and ascending and transverse colon before joining the splenic vein.
- **Inferior mesenteric vein (IMV):** Drains the descending colon, sigmoid colon, and proximal rectum before joining the splenic vein.
- **Internal iliac vein:** Drains the middle and distal rectum.
- **Middle rectal vein:** A branch of the internal iliac vein; drains upper anus.
- **Inferior rectal vein:** A branch of the internal pudendal vein; drains lower anus.
- **Hemorrhoidal complexes:** Three complexes within the anus that drain into the superior rectal veins.

WARD TIP

The ileocecal valve functions to prevent reflux of bowel contents from the cecum back to the ileum.

WARD TIP

The anatomy and physiology of the colon affect how colon cancers typically present. Left-sided colon cancers tend to present with a change in bowel habits (e.g., small-caliber stools, obstruction, and hematochezia). Right-sided colon cancers tend to present in a more indolent fashion with microcytic anemia, fatigue, and melena (dark, tarry stools) because the proximal colon has a larger circumference and the stool is less solid.

WARD TIP

The splenic flexure represents a "watershed" area between the areas supplied by the superior and inferior mesenteric arteries. This watershed area is particularly susceptible to ischemic injury as seen in ischemic colitis. The other two watershed zones are the ileocecal area and the junction of descending and sigmoid colon.

WARD TIP

The rectum has two major angles that play a significant role in continence. The first angle is formed at the origin of the rectum at the sacral promontory as it bends posteriorly and inferiorly, following the curve of the sacrum. The second angle, the anorectal angle, is formed by the puborectalis muscle as it joins the anus, pulling the rectum forward. A Valsalva maneuver enhances these angles, closing off the rectum.

Lymphatic Drainage

Lymphatics of the colon, rectum, and anus generally follow the arterial supply, with several levels of nodes as one moves centrally toward the aorta (e.g., ileocolic nodes, superior mesenteric nodes, etc.).

Innervation

- Derives primarily from autonomic nervous system.
- Sympathetic nerves: Inhibit peristalsis.
- Parasympathetic nerves: Stimulate peristalsis.

Histology

EXAM TIP

Anal canal above dentate line drains to inferior mesenteric nodes or to internal iliac nodes. Lower anal canal drains to inguinal nodes.

From inner lumen to outer wall:

- Colon: Mucosa, submucosa, inner circular muscle layer, outer longitudinal muscle (taenia coli).
- Rectum: Mucosa, submucosa, inner circular muscle, outer longitudinal muscle (confluent).
- Anus: Anoderm (epithelium that is richly innervated, but without secondary skin appendages).

Microbiology

- Colon sterile at birth; normal flora established shortly thereafter.
- Normal flora: 99% anaerobic (predominantly Bacteroides *fragilis*); 1% aerobic (predominantly Escherichia coli).

Physiology

General

The colon and rectum have three primary physiologic functions:

1. Absorption of water and electrolytes from stool
2. Storage of feces
3. Motility

EXAM TIP

The rectum can store approximately 500 cc of feces.

Motility

Characterized by three types of contractions:

1. Retrograde movements: From transverse colon to cecum, these movements slow the transit of luminal contents, thereby prolonging their exposure to absorptive epithelium.
2. Segmental contractions: The most common variety, these are localized simultaneous contractions of the longitudinal and circular muscles of the colon.
3. Mass movements:
   - Contractions of long segments of colon that are 30 seconds in duration and result in antegrade propulsion of luminal contents at a rate of 0.5–1 cm/sec.
   - Occur 3–4 times each day, especially after waking up or after eating, and may result in bowel movements.

- Neuronal control of colon:
  1. Extrinsic: Parasympathetic and sympathetic (as described above).
  2. Intrinsic (from mucosa to bowel wall): Mucosa, submucosal (Meissner's) plexus, circular muscle layer, myenteric (Auerbach's) plexus, longitudinal muscle layer, subserosal plexus, serosa.

defecation

1. Mass movement causes feces to move into rectal vault.
2. Sampling reflex: Rectal distention leads to involuntary relaxation of internal sphincter, allowing descent of rectal contents and sensation of feces at transitional zone.
3. Voluntary relaxation of external sphincter pushes contents down anal canal.
4. Voluntary increase in intra-abdominal pressure assists in propelling rectal contents out of anus.



WARD TIP

Constipation consists of the ability to pass flatus but the inability to pass stool.
obstipation refers to the inability to pass flatus or stool.

Disorders of Motility

irritable bowel syndrome (ibs)

definition

- Abnormal state of intestinal motility modified by psychosocial factors for which no anatomic cause can be found.
- IBS is often regarded as a wastebasket diagnosis for a change in bowel habits along with complaints of abdominal pain after other causes have been excluded.

Constipation

definition

< 3 stools/week.

diagnosis

By history; differentiate between acute constipation (persistent change in bowel habits for < 3 months), and chronic constipation (persistent change in bowel habits > 3 months). Constipation with absence of flatus is a hallmark of obstruction and is termed obstipation.

Common causes

Diet related (fluid, fiber), lack of physical activity, medications (especially opiates and anticholinergics), medical illness (IBS, diabetes, hypothyroidism), depression, neurologic disease (Parkinson's disease, multiple sclerosis), fecal impaction.

treatment

Depends on cause:

- Short-term: Stool softeners; enema if suppository fails. Fecal disimpaction (if present).
- Long-term: Encourage dietary changes (increasing fiber and fluid consumption).
- If dietary changes fail, assess colonic transit time. Defecography or anometry may prove helpful.

WARD TIP

Postvagotomy diarrhea occurs in 20% of patients after truncal vagotomy. Denervation of the extrahepatic biliary tree and small intestine leads to rapid transit of unconjugated bile salts into the colon, impeding water absorption and causing diarrhea. Cholestyramine is the medical treatment. If that fails, surgical reversal of a segment of the small intestine to prolong transit time and increase absorptive capacity may be considered.

DIARRHEA

Definition

- Passage of > 3 loose stools/day.
- A workup may be indicated to rule out infectious or ischemic etiologies.
- Diarrhea may occur due to extensive small bowel resection (short bowel syndrome), due to disruption of innervation, or even as an expected outcome (gastric bypass).

Diagnosis

- Stool sample for enteric pathogens and Clostridium difficile toxin.
- Check stool for white blood cells (WBCs) (IBD or infectious colitis), red blood cells (RBCs) without WBCs (ischemia, invasive infectious diarrhea, cancer). Fat content (steatorrhea, seen in chronic pancreatitis).
- Colonoscopy with biopsies in chronic diarrhea to rule out microscopic colitis.

Treatment

Individualized based on the treatable cause, and is addressed with the specific problems that may cause diarrhea (colitis, ischemia).

Colitis

PSEUDOMEMBRANOUS COLITIS

Definition

- An acute colitis characterized by formation of an adherent inflammatory exudate (pseudomembrane) overlying the site of mucosal injury. Most commonly due to overgrowth of C. difficile, a gram-positive, anaerobic, spore-forming bacillus.
- Typically occurs after broad-spectrum antibiotics (especially clindamycin, ampicillin, or cephalosporins) eradicate the normal intestinal flora.

Signs and symptoms

Vary from a self-limited diarrheal illness to invasive colitis with megacolon.

Diagnosis

Detection of C. difficile toxin in stool; proctoscopy or colonoscopy if diagnosis uncertain.

Treatment

Stop offending antibiotic; give flagyl or vancomycin PO (if patient unable to take PO, give flagyl IV).

Prognosis

High rate of recurrence (20%) despite high response rate to treatment.

WARD TIP

Patients with C. difficile colitis should be placed on contact isolation.

WARD TIP

Radiation-induced colitis can develop up to 20 years after radiation exposure and often results in an increased frequency of bowel movements for life.

RADIATION-INDUCED COLITIS

See Table 10-1.

- Associated with external radiation therapy (XRT) to pelvis usually for endometrial, cervical, prostate, bladder, and rectal cancer.
- **Risk factors:** Atherosclerosis, diabetes, hypertension, old age, adhesions from previous abdominal operation.

TABLE 10-1. Radiation-Induced Colitis

	Early (During Course of XRT)	Late (Weeks to years later)
Signs and symptoms	Nausea, vomiting, cramps, diarrhea, tenesmus, rectal bleeding	Tenesmus, bleeding, abscess, fistula involving rectum (rectal pain, stool per vagina)
Diagnosis	Plain abdominal films, barium enema	Barium enema, CT scan
Etiology	Mucosal edema, hyperemia, acute ulceration	Submucosal arteriolar vasculitis, microvascular thrombosis, wall thickening, mucosal ulceration, strictures, perforation
Treatment	Treat symptoms If no improvement, decrease dose of XRT or discontinue treatment	Treat with stool softener, topical 5-ASA, corticosteroid enema ▪ Strictures: Gentle dilatation or diverting colostomy after excluding cancer ▪ Rectovaginal fistula: Proximal colostomy and low colorectal anastomosis or coloanal temporary colostomy

- Chance of developing disease is dose dependent:
  < 4,000 cGy No patients
  5,000–6,000 cGy Some patients
  > 6,000 cGy Most patients

Ischemic Colitis

Definition

Acute or chronic intestinal ischemia secondary to decreased intestinal perfusion or thromboembolism:

- Embolus or thrombus of the IMA.
- Poor perfusion of mucosal vessels from arteriole shunting or spasm.

Incidence

Most common in the elderly.

Risk Factors

- Old age
- Status post abdominal aortic aneurysm (AAA) repair
- Hypertension
- Coronary artery disease, atrial fibrillation
- Cocaine abuse
- Prothrombotic conditions
- Sickle cell anemia
- Unaccustomed exertion

WARD TIP

Ischemic colitis often affects the splenic flexure.

EXAM TIP

typical scenario: A 70-year-old white male with a history of hypertension develops cramping lower abdominal pain 1 day s/p AAA repair. A few hours later he develops bloody diarrhea. What's the diagnosis? Think: Ischemic colitis should be suspected in any elderly patient who develops acute abdominal pain followed by rectal bleeding. Furthermore, the most common setting for ischemic colitis is the early postoperative period after AAA repair when impaired blood flow through the IMA may put the colon at risk.

Signs and Symptoms

- Mild lower abdominal pain and rectal bleeding, classically after AAA repair.
- Pain more insidious in onset than small bowel ischemia.

diagnosis

- Clinical history.
- Plain abdominal x-ray—may reveal pneumatosis (air in bowel wall) or "thumbprinting" (submucosal edema).
- Computed tomographic (CT) scan of the abdomen may reveal segmental thickening of bowel wall.
- Colonoscopy may show pale mucosa with petechial bleeding.

treatment

If symptoms are mild, administer IV fuids and observe; if moderate (with fever and increased WBC), give IV antibiotics; if severe (with peritoneal signs), exploratory laparotomy with colostomy.

inflAmmAtory bowEl diseAsE: ulCErAtivE Colitis

definition

Inflammation confined to mucosal layer of colon that extends from the rectum proximally in a continuous fashion. This is an autoimmune process.

Incidence

Age between 15 and 40 or 50 and 80 (bimodal age distribution).

- Whites 4× > nonwhites
- Industrialized nations >> developing nations

Risk Factors

- Jewish descent
- White race
- Urban dwelling
- Positive family history
- Nicotine decreases risk (unlike Crohn's disease)

WARD TIP

Both ulcerative colitis (UC) and Crohn's disease can present with bloody diarrhea, but it is more common in UC.

Signs and Symptoms

- Mild disease (confined to rectum or rectosigmoid): Intermittent rectal bleeding, passage of mucus from rectum, mild diarrhea.
- Moderate disease: Frequent loose bloody stools, mild abdominal pain, low-grade fever.
- Severe disease: Frequent loose stools, severe abdominal pain, bleeding necessitating blood transfusion. Patients may have rapid weight loss.

diagnosis

- Flexible sigmoidoscopy with histolopathologic evaluation of biopsies.
- Barium enema: "Lead pipe" appearance of colon due to loss of haustral folds, but no longer test of choice.

treatment

- Medical: Similar to Crohn's (see Table 10-2):
  - Mild/moderate disease: 5-ASA, corticosteroids PO or per rectum.
  - Severe disease: IV steroids.
  - Proctitis: Topical steroids.
  - Refractory disease: Immunosuppression.

TABLE 10-2. Ulcerative Colitis vs. Crohn's disease

	ULCERATIVE COLITIS	CROHN'S DISEASE
Pathology	▪ Inflammation of the mucosa only (exudate of pus, blood, and mucus from the "crypt abscess") ▪ Always starts in rectum (up to one third don't progress) ▪ Limited to colon and rectum	▪ Inflammation involves all bowel wall layers, which is what may lead to fistulas and abscess ▪ Rectal sparing in 50% ▪ May affect mouth to anus
Diagnosis	▪ Continuous lesions ▪ Rare ▪ Lead pipe colon appearance due to chronic scarring and subsequent retraction and loss of haustra	▪ Skip lesions: Interspersed normal and diseased bowel ▪ Aphthous ulcers ▪ Cobblestone appearance from submucosal thickening interspersed with mucosal ulceration
Complications	▪ Perforation ▪ Stricture ▪ Megacolon ▪ Cancer	▪ Abscess ▪ Fistulas ▪ Obstruction ▪ Cancer ▪ Perianal disease

- Surgical:
  - Indications: Failure of medical therapy, increasing risk of cancer in long-standing disease, bleeding, perforation.
  - Procedure: Proctocolectomy with end ileostomy or restorative J-pouch creation (curative).
  - If patient is acutely ill and unstable due to perforation, a diverting loop colostomy is indicated. Once stabilized, the patient may undergo a more definitive operation.
  - In Crohn's disease the treatment is stricturoplasty or segmental resections because recurrence is the rule and the goal is to preserve as much healthy intestine as possible.

Prognosis

Approximately 1–2% risk of cancer at 10 years, and 1%/year thereafter.

EXAM TIP

Unless all the colonic and rectal mucosa is removed in ulcerative colitis, the patient is still at risk for cancer.

EXAM TIP

Patients with UC need colonoscopy 10 years after diagnosis to screen for CRC.

Diverticular Disease

Definition

- Herniation of the mucosa through the muscular layers of the bowel wall at sites where arterioles penetrate, forming small outpouchings or diverticula.
- Are generally numerous, collectively referred to as **diverticulosis**.
- **Diverticulitis** refers to the inflammation of diverticula.

Incidence

- > 50% of Americans over 70 years of age.
- Men and women equally affected.

- Sigmoid colon most commonly involved with progressively decreasing frequency of involvement as one proceeds proximally.

risk factors

- Old age
- Low-fiber diet

signs And symptoms

Diverticulosis

- Eighty percent of patients are asymptomatic.
- **Massive, painless lower GI bleeding is classic** (notably absent in diverticulitis).

Diverticulitis

- Persistent abdominal pain often localized to the left lower quadrant (LLQ) with development of peritoneal signs as the disease progresses.
- LLQ and/or pelvic tenderness.
- Ileus/abdominal distention.
- Anorexia, nausea, vomiting, and change in bowel habits.
- Fever.
- Elevated WBC.

diAgnosis

Diverticulosis

- Characteristic history and physical exam confirmed by diverticula identified on CT/barium enema and/or colonoscopy.
- Treatment: High-fiber diet.
- See section on lower GI bleed (LGIB) for management of patients who present with acute LGIB.

Diverticulitis

- Characteristic history and physical exam.
- Elevated WBCs.
- CT scan (test of choice): Pericolonic inflammation with or without abscess formation.
- Barium enema and colonoscopy may induce perforation and are contraindicated in the acute setting but should be obtained in follow-up (see Figure 10-2).
- Abdominal x-ray: Ileus, distention, and/or free intraperitoneal air.

EXAM TIP

The diverticula of common diverticulosis are false diverticula; all the layers of the bowel wall would need to herniated to be a "true" diverticula.

EXAM TIP

Any generalized peritonitis in diverticulitis means it should be classified as at least Hinchey III or IV.

EXAM TIP

Pathophysiology of diverticulitis: A peridiverticular inflammation caused by microperforation of the diverticulum. Feces extravasate onto the serosal surface but infection is usually well contained in a patient with normal immune function.

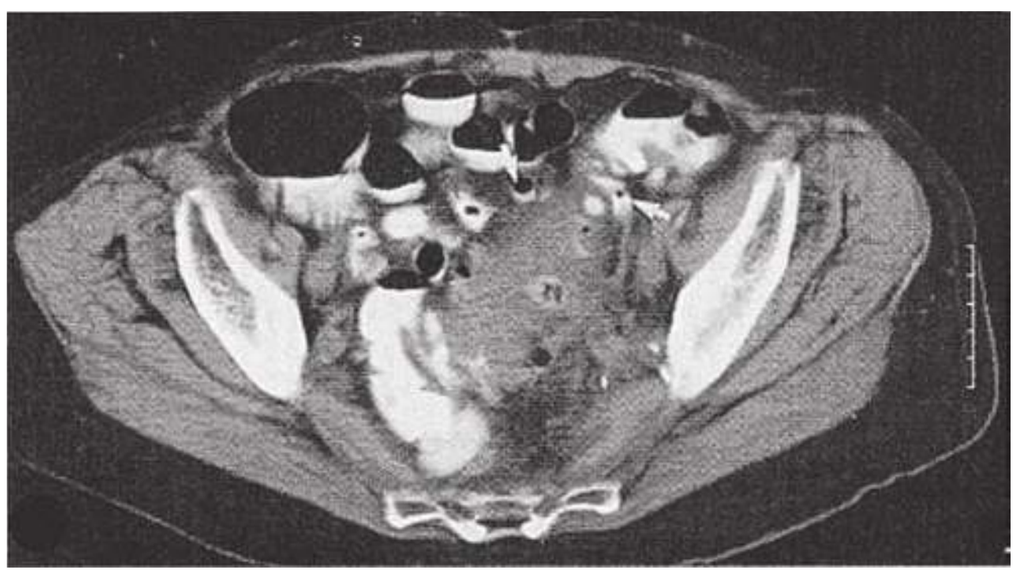


FIGURE 10-2. Ct scan demonstrating multiple small sigmoid diverticuli (arrows).
(Reproduced, with permission, from Gupta H, Dupuy DE. Surg Clin North Am 6(77); December 1997.)

Treatment

- **Uncomplicated diverticulitis:**
  - Outpatient management: Clear liquid diet, PO antibiotics, and non-opioid analgesics with close follow-up.
  - Follow-up includes colonoscopy and dietary recommendations once acute infection has subsided.
  - If outpatient therapy fails, admit for IV antibiotics and IV hydration with bowel rest. Nasogastric tube (NGT) is placed when there is evidence of ileus or small bowel obstruction (SBO), with nausea and vomiting.
- The Hinchey staging system is often used to describe the severity of **complicated diverticulitis:**
  - Stage I includes colonic inflammation with an associated pericolic abscess.
  - Stage II includes colonic inflammation with a retroperitoneal or pelvic abscess.
  - Stage III is associated with purulent peritonitis.
  - Stage IV is associated with fecal peritonitis.
    - Stage I and II are treated by IV antibiotics and CT-guided aspiration.
  - If the abscess is inaccessible to drainage and not responding to antibiotics, then it is treated surgically (drainage with Hartmann pouch or sigmoid colectomy).
    - Stage III and IV need operative management.
  - In the emergent setting, a Hartmann's procedure is performed.
  - Elective resection of affected bowel may be considered in the patient who has recurrent episodes of diverticulitis requiring treatment or complications of diverticulitis.
  - All patients with diverticulitis should undergo a full colonoscopy 4–6 weeks after the attack to rule out malignancy, as sometimes colon cancer mimics diverticulitis.

Prognosis

One third of patients remain asymptomatic, one third have episodic pain, and one third progress to have a recurrence. Chronic complications include stricture, colovesical/colovaginal fistula.

Lower GI Bleed (LGIB)

Definition

- GI bleeding distal to the ligament of Treitz.
- LGIB is considered massive when the patient requires 3 or more units of blood within 24 hours.
- Most common causes are diverticulosis and angiodysplasia. Other causes include cancer, IBD, ischemic colitis, hemorrhoids.
- Anticoagulation treatment increases the risk for LGIB.

Management

See Table 10-3.

WARD TIP

Ten to 25% of LGIBs eventually require surgery, despite the fact that 85% of bleeds initially stop spontaneously.

WARD TIP

An extensive operation is indicated when no site is identified in an unstable patient because although < 10% will rebleed, the mortality for those 10% is approximately 30%.

EXAM TIP

In a patient presenting with LGIB and a mid-systolic murmur in the right second intercostal space, think **Heyde syndrome**—angiodysplasia (causing bleeding from colon) with aortic stenosis.

TABLE 10-3. management of lower GI Bleeding (lGIB)

	Diverticulosis	angiodysplasia
Incidence	50% of patients are > 60	25% of patients are > 60; men > women
Character	Painless, > 60% site of bleeding proximal to splenic flexure	Cecum and ascending colon
Quantity and rate	Massive and rapid	Slow
Signs and symptoms	Melena and/or hematochezia often with symptoms of orthostasis	
Diagnosis	▪ First rule out upper GI bleeding with nasogastric lavage ▪ To identify site of bleed: 1. Colonoscopy 2. ≥ 0.5 mL/min: Bleeding scan with Tc-sulfur colloid identifies bleeding; label lasts for up to 24 hours so a patient can be easily rescanned when rebleeding occurs after a negative initial scan 3. ≥ 1 mL/min: Angiography (selective mesenteric angiography best method to diagnose angiodysplasia)	
Treatment	1. Resuscitation 2. Therapeutic options if site identified: Octreotide, embolization, vasoconstriction with epinephrine, vasodestruction with alcohol or sodium compounds, or coagulation/cautery with heat 3. If site identified but bleeding massive or refractory, segmental colectomy 4. Without identification of bleeding site and persistent bleeding in an unstable patient, an exploratory laparotomy with possible total abdominal colectomy with ileostomy	
Cause	Disruption of arteriole at either dome or antimesenteric neck of diverticulum (almost always on mucosal side, so bleed occurs into lumen rather than into peritoneal cavity)	Chronic intermittent obstruction of submucosal veins secondary to repeated muscular contractions results in dilated venules with incompetent precapillary sphincters and thus arteriovenous communication
Prognosis	10% overall mortality	

Large Bowel Obstruction

incidence

Most commonly occurs in elderly patients; much less common than SBO.

signs And symptoms

Abdominal distention, cramping abdominal pain, nausea, vomiting, obstipation.

diAgnosis

- Supine and upright abdominal films: Distended proximal colon, air-fluid levels, and no distal rectal air.
- Establish 8- to 12-hour history of obstipation; passage of some gas or stool indicates partial bowel obstruction.
- Barium enema: May be necessary to distinguish between ileus and obstruction.

WARD TIP

The three most common causes of obstruction of the large bowel are adenocarcinoma (65%), scarring secondary to diverticulitis (20%), and volvulus (5%).

Treatment

1. Correction of fluid and electrolyte abnormalities.
2. Nasogastric tube for intestinal decompression (as gastric emptying is reflexly inhibited).
3. Broad-spectrum IV antibiotics (e.g., cefoxitin).
4. Relieve obstruction surgically (colonic obstruction is a surgical emergency since a nasogastric tube will not decompress the colon).

Volvulus

Definition

Rotation of a segment of intestine about its mesenteric axis; characteristically occurs in the sigmoid colon (75% of cases) or cecum (25%).

Incidence

More than 50% of cases occur in patients over 65.

Risk Factors

- Elderly (especially institutionalized patients).
- Chronic constipation.
- Pyschotropic drugs.
- Hypermobile cecum secondary to incomplete fixation during intrauterine development (cecal volvulus).

Signs And Symptoms

See Large Bowel Obstruction.

Diagnosis

- Clinical presentation.
- Abdominal films: Markedly dilated sigmoid colon or cecum with a "kidney bean" appearance.
- Barium enema: Characteristic "bird's beak" at areas of colonic narrowing.

Treatment

- Cecal volvulus: Right hemicolectomy.
- Sigmoid volvulus (see Figure 10-3):
  - Sigmoidoscopy with rectal tube insertion to decompress the volvulus.
  - Emergent laparotomy if sigmoidoscopy fails or if strangulation or perforation is suspected.
  - Elective resection in same hospital admission to prevent recurrence (nearly 50% of cases recur after nonoperative reduction).



FIGURE 10-3. Abdominal x-ray shows a grossly distended coffee bean–shaped loop of bowel in the right upper quadrant, findings that are typical of a sigmoid volvulus. (Reproduced, with permission, from Rozycki GS, Tremblay L, Feliciano DV, et al. Three Hundred Consecutive Emergent Celiotomies in General Surgery Patients: Influence of Advanced Diagnostic Imaging Techniques and Procedures on Diagnosis. Annals of Surgery 2002: 235(5); 681–689.)

Pseudo-obstruction (Ogilvie Syndrome)

Definition

Massive colonic dilation without evidence of mechanical obstruction.

Incidence

More common in older, institutionalized patients.

Risk Factors

- Severe infection, recent surgery or trauma.
- Polypharmacy, particularly antipsychotics.

Signs And Symptoms

Marked abdominal distention with mild abdominal pain and decreased or absent bowel sounds.

Diagnosis

- Abdominal radiograph with massive colonic distention.
- Exclude mechanical cause for obstruction with water-soluble contrast enema and/or colonoscopy.

Treatment

- NGT and rectal tube for proximal and distal decompression, respectively.
- Correction of electrolyte abnormalities.

WARD TIP

Ogilvie syndrome is associated with any severe acute illness, neuroleptics, opiates, malignancy, and certain metabolic disturbances.

WARD TIP

In Ogilvie syndrome, pharmacologic decompression of the bowel with neostigmine is particularly useful but should be performed in a monitored setting. Do not give neostigmine if bradycardic!

- Discontinue narcotics, anticholinergics, or other offending medications.
- Consider pharmacologic decompression with neostigmine. Neostigmine is a cholinesterase inhibitor and is contraindicated in patients with cardiac disease.
  - Colonoscopic decompression is an alternative to neostigmine with similar success.
- If peritoneal signs develop, the patient should undergo prompt exploratory laparotomy to treat possible perforation.
- Refractory cases may need total colectomy.

Benign Tumors of the Large Bowel

Colorectal polyps

morphology

Can be classified into sessile (flat) and pedunculated (on a stalk).

Histologic types

- Inflammatory (pseudopolyp): Seen in UC.
- Lymphoid: Mucosal bumps containing intramucosal lymphoid tissue; no malignant potential.
- Hyperplastic: Overgrowth of normal tissue; no malignant potential.
- Adenomatous: Premalignant; are classified as tubular (75%), tubulovillous (15%), and villous (10%).
- Hamartomatous: Normal tissue arranged in abnormal configuration; juvenile polyps, Peutz–Jeghers polyps.

Incidence

Thirty to 40% of individuals over 60 in the United States.

Signs and Symptoms

- Asymptomatic (most common)
- Melena
- Hematochezia
- Mucus
- Change in bowel habits

diagnosis

Flexible endoscopy (sigmoidoscopy or colonoscopy).

treatment

- Colonoscopic resection.
- If colonoscopic resection not possible a segmental colon resection is indicated.

polyposis syndromEs

See Table 10-4.

WARD TIP

Malignant potential of a polyp is determined by: size, histologic type, and epithelial dysplasia.

SIZE	RISK OF CA
< 1 cm	1–3%
1–2 cm	10%
> 2 cm	40%
HISTOLOGY	**RISK**
Tubular	5%
Tubulovillous	20%
Villous	40%
ATYPIA	**RISK**
Mild	5%
Moderate	20%
Severe	35%

TABLE 10-4. Polyposis Syndromes of the Bowel

SYNDROME	INHERITANCE PATTERN	RISKS/ASSOCIATED FINDINGS
Familial polyposis coli (FAP)	Autosomal dominant	▪ Hundreds to thousands of polyps develop between the second and fourth decades; colon cancer inevitable without prophylactic colectomy ▪ Caused by abnormal gene on chromosome 5, APC gene ▪ Indication for operation: Polyps ▪ Operations: 1. Proctocolectomy with ileostomy 2. Colectomy with ileorectal anastomosis 3. Proctocolectomy with ileal pouch—anal anastomosis
Gardner's syndrome	Autosomal dominant	Innumerable polyps with associated osteomas, epidermal cysts, and fibromatosis; colon cancer inevitable without surgery
Turcot's syndrome	Autosomal dominant	Multiple adenomatous colonic polyps with central nervous system (CNS) tumors (especially gliomas)
Peutz–Jeghers syndrome	Autosomal dominant	Hamartomatous polyps of the entire GI tract with melanotic pigmentation of face, lips, oral mucosa, and palms; increased risk for cancer of the pancreas, cervix, lung, ovary, and uterus
Hereditary nonpolyposis colon cancer syndrome (HNPCC or Lynch syndrome)	Autosomal dominant	Lynch syndrome I: Patients without multiple polyps who develop predominantly right-sided colon cancer at a young age. Lynch syndrome II: Same as Lynch I but additional risk for extracolonic adenocarcinomas of the uterus, ovary, cervix, and breast

Colorectal Carcinoma (CRC)

INCIDENCE

- Second most common cause of cancer deaths overall (behind lung cancer).
- 130,000 new cases and 55,000 deaths each year.
- Incidence increases with increasing age starting at age 40 and peaks at 60–79 years of age.
- See Table 10-5 for screening recommendations from the U.S. Preventative Services Task Force.

EXAM TIP

At diagnosis of CRC: 10% in situ disease; one third local disease; one third regional disease; 20% metastatic disease.

RISK FACTORS

- Age > 50.
- Personal history of resected colon cancer or adenomas.
- Family history of colon cancer or adenomas.
- Low-fiber, high-fat diet.
- Inherited colorectal cancer syndrome (familial adenomatous polyposis [FAP], hereditary nonpolyposis colon cancer [HNPCC]).
- Long-standing UC or Crohn's disease.

TABLE 10-5. Screening Guidelines for Colorectal Cancer

POPULATION	INITIAL AGE	RECOMMENDED SCREENING TEST
Average risk	50 years	Annual FOBT or Flexible sigmoidoscopy every 5 years or Annual FOBT and flexible sigmoidoscopy every 5 years or Air-contrast barium enema every 5 years or Colonoscopy every 10 years
Adenomatous polyps	50 years	Colonoscopy at first detection; then colonoscopy in 3 years If no further polyps, colonoscopy every 5 years If polyps, colonoscopy every 3 years Annual colonoscopy for > 5 adenomas
Colorectal cancer	At diagnosis	Pretreatment colonoscopy; then at 12 months after curative resection; then colonoscopy after 3 years; then colonoscopy every 5 years, if no new lesions
Ulcerative colitis, Crohn's colitis	At diagnosis; then after 8 years for pacolitis, after 15 years for left-sided colitis	Colonoscopy with multiple biopsies every 1–2 years
FAP	10–12 years	Annual flexible sigmoidoscopy Upper endoscopy every 1–3 years after polyps appear
Attenuated FAP	20 years	Annual flexible sigmoidoscopy Upper endoscopy every 1–3 years after polyps appear
HNPCC	20–25 years	Colonoscopy every 1–2 years Endometrial aspiration biopsy every 1–2 years
Familial colorectal cancer first-degree relative	40 years or 10 years before the age of the youngest affected relative	Colonoscopy every 5 years Increase frequency if multiple family members are affected, especially before 50 years

FAP = familial adenomatous polyposis; FOBT = fecal occult blood testing; HNPCC = hereditary nonpolyposis colon cancer.

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Table 29-2.

ADENOMA-CARCINOMA SEQUENCE

Normal → hyperproliferative → early adenoma → intermediate adenoma → late adenoma → carcinoma (→ metastatic disease).

1. APC gene loss or mutation
2. Loss of DNA methylation
3. Ras (gene) mutation
4. Loss of DCC gene
5. Loss of p53 gene

Signs and Symptoms

- Typically asymptomatic for a long period of time; symptoms, if present, depend on location and size.

WARD TIP

Rule out metastases from colorectal cancer with chest CT, CT of abdomen and pelvis. Measure carcinoembryonic antigen (CEA) to establish a baseline level.

- Right-sided cancers: Occult bleeding with melena, anemia, and weakness.
- Left-sided cancers: Rectal bleeding, obstructive symptoms, change in bowel habits and/or stool caliber.
- Both: Weight loss, anorexia.

diagnosis

- Colon cancer: Flexible sigmoidoscopy or colonoscopy (need to evaluate entire colon and rectum to look for synchronous lesions).
- Rectal cancer: Digital rectal exam, proctoscopy/colonoscopy, barium enema, also consider transrectal ultrasound (TRUS), CT, or magnetic resonance imaging (MRI) to assess depth of local tumor invasion and local lymph node status.

WARD TIP

Microcytic anemia in an elderly male or postmenopausal woman is colon cancer until proven otherwise.

Staging and Prognosis

TNM System (more current system)

Tx: Primary tumor cannot be assessed
T0: No evidence of primary tumor
Tis: Carcinoma in situ: intraepithelial or invasion of lamina propria
T1: Invasion of submucosa
T2: Invasion of muscularis propria
T3: Invasion of subserosa, or nonperitonealized pericolic or perirectal tissues
T4: Invasion of visceral peritoneum/direct invasion of other organs
Nx: Regional lymph nodes cannot be assessed
N0: No nodal disease
N1: 1–3 pericolic or perirectal lymph nodes
N2: 4 or more lymph nodes
M0: No evidence of distant metastasis
M1: Distant metastasis

EXAM TIP

CRC 5-year survival by stage:
Stage I: T1N0M0/T2N0M1 (90%)
Stage II: T3N0M0/T4N0M0 (75%)
Stage III: any T/**n1**M0/any TN2–3M0 (50%)
Stage IV: any T any N **m1** (5%)

treatment

- Surgical resection (see Table 10-6 and Figure 10-5):
  - Goal is to remove primary tumor along with lymphatics draining involved bowel.
  - In rectal cancer, the circumferential radial margin (CRM) is crucial to local recurrence. Total mesorectal excision (TME) reduces the rates of local recurrence.
- Adjuvant treatment:
  - Stage III: 5-fluorouracil (5-FU)-based chemotherapy.
  - Rectal cancer: Preop radiation using 5-FU as a radiosensitizer (this sequence is called "neoadjuvant" therapy because it occurs prior to the definitive surgical treatment).

EXAM TIP

typical scenario: A 65-year-old male presents complaining of rectal bleeding and increasing constipation with thinning of stool caliber. A barium enema is performed. What is the most likely diagnosis? Think: This elderly individual is at risk for colon cancer, and his obstructive symptoms likely indicate that it is left-sided. The "apple core" filling defect in the descending colon on barium enema is classic for left-sided colon cancer. See Figure 10-4.

WARD TIP

There is no classification system for external hemorrhoids. They are either present or absent.

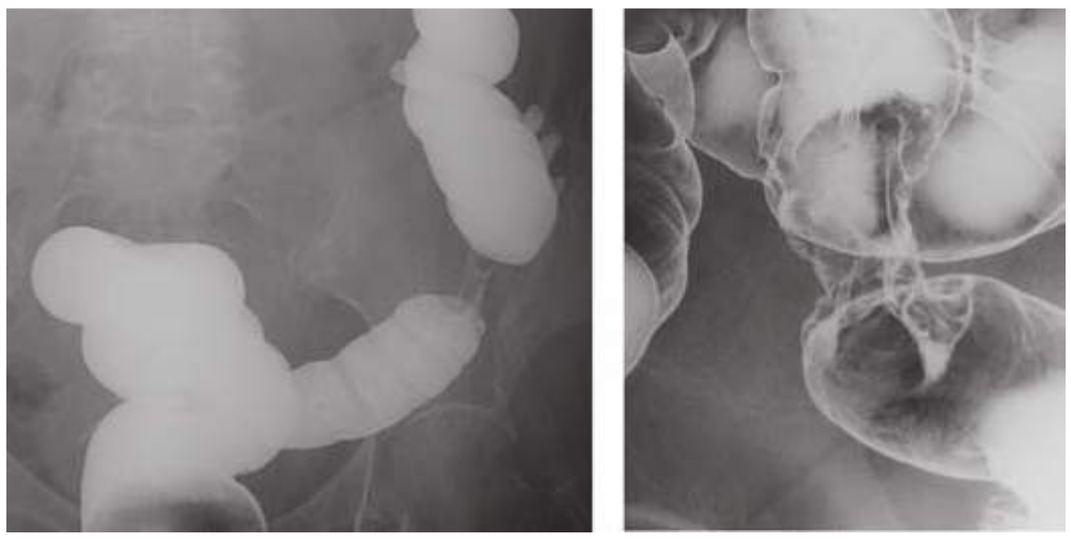


FIGURE 10-4. apple core lesions. (Reproduced, with permission, from Kaiser AM. McGraw-Hill Manual: Colorectal Surgery. New York, NY: McGraw-Hill Education; 2009. Figure 2-4B.)

TABLE 10-6. Operative management of CRC Based on tumor location

TUMOR LOCATION	OPERATION
Cecum	Right hemicolectomy: ▪ Resection of terminal ileum, cecum, ascending and proximal transverse colon
Right colon	Right hemicolectomy
Proximal/mid-transverse colon	Extended right hemicolectomy: ▪ Resection as above plus remainder of transverse colon and splenic flexure
Splenic flexure and left colon	Left hemicolectomy: ▪ Resection of distal transverse, descending and sigmoid colon
Sigmoid or rectosigmoid colon	Sigmoid colectomy
Proximal rectum	Low anterior resection (LAR): ▪ Tumors > 4 cm from anal verge ▪ Must be able to get 2-cm margin ▪ Includes total mesorectum excision ▪ Involves complete mobilization of rectum, with division of lateral ligaments, posterior mobilization through Waldeyer's fascia to tip of coccyx, dissection between rectum and vagina or prostate ▪ Complications: Incontinence, urinary dysfunction, sexual dysfunction, anastomotic leak (5–10%), stricture (5–20%)
Distal rectum	Abdominal-perineal resection (APR): ▪ Tumors too low for LAR (less than 2 cm from anorectal ring) ▪ Involves creation of end ostomy, with resection of rectum, total mesorectal excision (TME), and closure of anus ▪ Complications: Stenosis, retraction or prolapse of ostomy, perineal wound infection
Other situations	▪ Obstructing cancer: Attempt to decompress ▪ Perforated cancer: Remove disease and perforated segments ▪ Synchronous or metachronous lesions, or proximal perforation with distal cancer: Subtotal colectomy with ileosigmoid or ileorectal anastomosis ▪ Very distal rectal tumor and/or patient not stable for big operation: Transanal excision of tumor, endoscopic microsurgery, or endocavitary radiation ▪ En-bloc resection for malignant fistulas

Perianal and Anal Problems

Hemorrhoids

See Table 10-7.

- Prolapse of the submucosal veins located in the left lateral, right anterior, and right posterior quadrants of the anal canal.
- Classified by type of epithelium: Internal if covered by columnar mucosa (above dentate line), external if covered by anoderm (below dentate line), and mixed if both types of epithelia are involved.
- **Incidence:** Male = female.

EXAM TIP

Above the dentate line there are no pain sensory fibers, as such internal hemorrhoids above this line are painless.

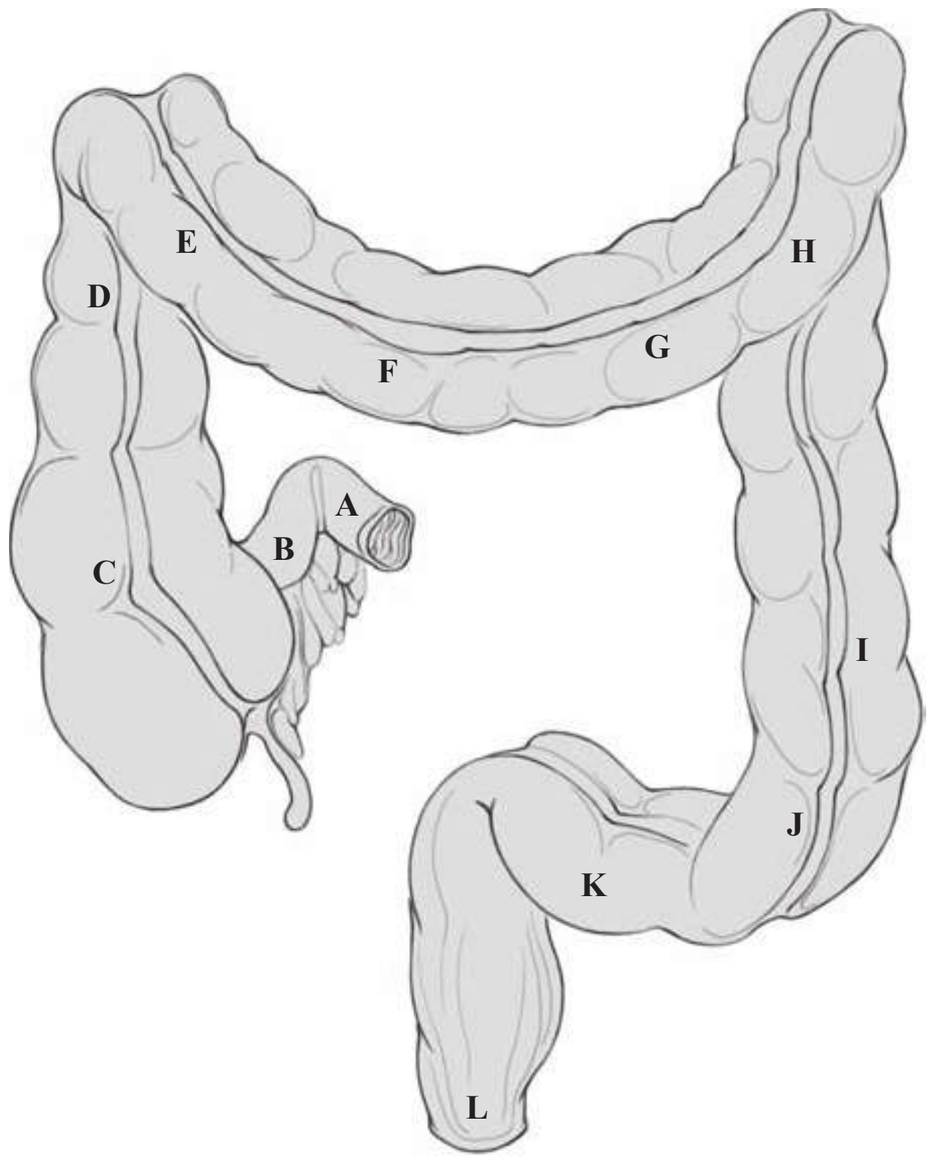


FIGURE 10-5. Terminology of types of colorectal resections. A to C, ileocecectomy; + A + B to D, ascending colectomy; + A + B to F, right hemicolectomy; + A + B to G, extended right hemicolectomy; + E + F to G + H, transverse colectomy; G to I, left hemicolectomy; F to I, extended left hemicolectomy; J + K, sigmoid colectomy; + A + B to J, subtotal colectomy; + A + B to K, total colectomy; + A + B to L, total proctocolectomy.

TABLE 10-7. Grade Description, Symptoms, and Treatments of Internal Hemorrhoids

	GRADE DESCRIPTION	SYMPTOMS	TREATMENT
I	Protrudes into lumen, no prolapse	Bleeding	Nonresectional measures[a]
II	Prolapse with straining, spontaneous return	Bleeding, perception of prolapse	Nonresectional measures
III	Prolapse, requires manual reduction	Bleeding, prolapse, mucous soilage, pruritus	Consider trial of nonresectional measures; many require excision
IV	Prolapse cannot be reduced	Bleeding, prolapse, mucous soilage, pruritus, pain[b]	Excision

[a]Nonresectional methods (rubber-band ligation, infrared coagulation, or injection sclerotherapy) can be used in insensate tissue only (above the dentate line).

[b]Pain if thrombosed or ischemic.

Reproduced, with permission, from Niederhuber JE. Fundamentals of Surgery. Stamford, CT: Appleton & Lange; 1998: 317.

- **Risk factors:** Constipation, pregnancy, increased pelvic pressure (ascites, tumors), portal hypertension.
- **Diagnosis:** Clinical history, physical exam, visualize with anoscope.

Anal fissure

definition

Painful linear tears in the anal mucosa below the dentate line; induced by constipation or excessive diarrhea.

Signs and Symptoms

- Pain with defecation.
- Bright red blood on toilet tissue.
- Markedly increased sphincter tone and extreme pain on digital examination.
- Visible tear upon gentle lateral retraction of anal tissue.

diagnosis

History and physical exam.

treatment

- Sitz baths.
- Fiber supplements, bulking agents.
  - Medical management with stool softeners, nitropaste, Botox, or diltiazem.
- Increased fluid intake.
  - Topical nifedipine or nitroglycerine ointment.
  - Chemodenervation of the internal sphincter with Botulinum toxin injection.
- Lateral internal sphincterotomy.

Anorectal abscess

definition

Obstruction of anal crypts with resultant bacterial overgrowth and abscess formation within the intersphincteric space.

Risk Factors

- Constipation/diarrhea/IBD.
- Immunocompromise.
- History of recent surgery or trauma.
- History of CRC.
- History of previous anorectal abscess.

Signs and Symptoms

Rectal pain, often of sudden onset, with associated fever, chills, malaise, leukocytosis, and a tender perianal swelling with erythema and warmth of overlying skin.

treatment

Surgical drainage.

EXAM TIP

typical scenario: A 24-year-old male with chronic constipation complains of intense anal pain. He has a tender, swollen, bluish lump at the anal orifice. What is the treatment? Think: This young man's history of constipation and physical exam findings are classic for a thrombosed external hemorrhoid. The treatment is surgical excision if the pain has been present for < 48 hours or is persistent. Pain typically subsides after 48 hours, and treatment is symptomatic.

EXAM TIP

- Anal fissures are always posterior and midline.

Anorectal fistulas

WARD TIP

Goodsall's rule (Figure 10-6) can be used to clinically predict the course of an anorectal fistula tract. Imagine a line that bisects the anus in the coronal plane. Any fistula that originates anterior to the line will course anteriorly in a direct route. Fistulae that originate posterior to the line will have a curved path.

definition

Tissue tracts (abnormal connections between two areas) originating in the glands of the anal canal at the dentate line that are usually the chronic sequelae of anorectal infections, particularly abscesses.

Classification of anorectal Fistulas

1. **Intersphincteric (most common):** Fistula tract stays within intersphincteric plane.
2. **Transsphincteric:** Fistula connects the intersphincteric plane with the ischiorectal fossa by perforating the external sphincter.
3. **Suprasphincteric:** Similar to transsphincteric, but the fistula loops above the external sphincter to penetrate the levator ani muscles.
4. **Extrasphincteric:** Fistula passes from rectum to perineal skin without penetrating sphincteric complex.

Signs and Symptoms

Recurrent or persistent perianal drainage that becomes painful when one of the tracts becomes occluded.

diagnosis

- Digital rectal exam.
- Anoscopy.
- If the internal opening cannot be identified by direct probing, it should be identified by probing the external opening or by injecting methylene blue or hydrogen peroxide into the tract.

treatment

Intraoperative unroofing of the entire fistula tract with or without placement of setons (vessel loop placed in the fistula tract to keep it patent for drainage).

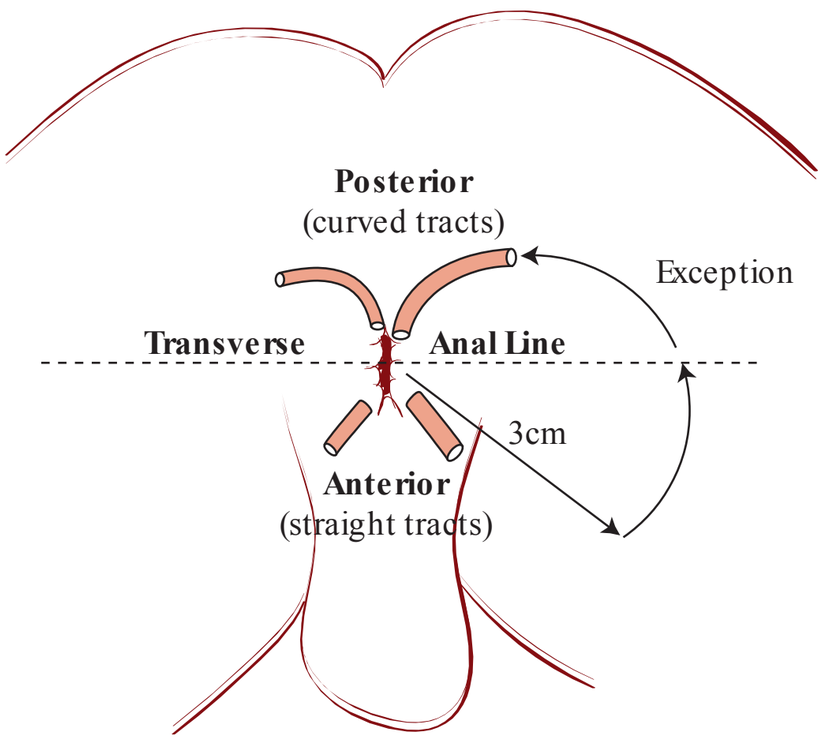


FIGURE 10-6. Goodsall's rule to identify the internal opening of fistulas-in-ano.
(Reproduced, with permission, from Brunicardi FC, Andersen Dk, Billiar TR, et al., eds. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2005: 1108.)

Pilonidal Disease

Definition

A cystic inflammatory process generally occurring at or near the cranial edge of the gluteal cleft.

Incidence

Most commonly seen in hirsute men in their late teens to the third decade.

Signs and Symptoms

Can present acutely as an abscess (fluctuant mass) or chronically as a draining sinus with pain at the top of the gluteal cleft.

Treatment

- Acute presentation: incision and drainage under local anesthesia with removal of involved hairs.
- Chronic disease: Hair removal and observation or excision.

WARD TIP

The terms pilo and nidus are Latin terms for "hair" and "origin," respectively, indicating that the lesion is associated with hair follicles. Pilonidal disease results from trauma to hair follicles in the gluteal region with resultant infection.

Anal Cancer

Definition

Neoplasms of the anorectal region that are classified into tumors of the perianal skin (anal margin carcinomas) and tumors of the anal canal.

Incidence

Rare (1–2% of all colon cancers).

Risk Factors

- Human papillomavirus (HPV)
- Human immunodeficiency virus (HIV)
- Cigarette smoking
- Multiple sexual partners
- Anal intercourse
- Immunosuppressed state

WARD TIP

Two unique in situ tumors of the perianal skin are Paget's disease and Bowen's disease. Paget's disease of the anus is adenocarcinoma in situ, and anal Bowen's disease is squamous carcinoma in situ.

Signs And Symptoms

Often asymptomatic; can present with anal bleeding, a lump, or itching; an irregular nodule that is palpable or visible externally (anal margin tumor) or a hard, ulcerating mass that occupies a portion of the anal canal (anal canal tumor).

Diagnosis

- Surgical biopsy with histopathologic evaluation.
- **Histology:** Anal margin tumors include squamous and basal cell carcinomas, Paget's disease, and Bowen's disease. Anal canal tumors are usually

epidermoid (squamous cell carcinoma or transitional cell/cloacogenic carcinoma) or malignant melanoma.
- **Clinical staging:** Involves history, physical exam, colonoscopy, abdominal or pelvic CT or MRI, CXR, and liver function tests.

treatment

- Epidermoid carcinoma of anal canal: Chemoradiation is mainstay—5-FU, mitomycin C, and external beam radiation (Nigro protocol) surgery is reserved for recurrence.
- Other anal margin tumors: Wide local excision alone or in combination with radiation and/or chemotherapy is successful in 80% of cases without abdominal-perineal resection (APR) if tumor is small and not deeply invasive.
- Anal canal tumors: Local excision not an option; combined chemotherapy (5-FU and mitomycin C) with radiation often successful; APR if chemoradiation fails.

prognosis

- Anal margin tumors: 80% overall 5-year survival.
- Anal canal tumors:
  - Epidermoid carcinoma: 50% overall 5-year survival.
  - Malignant melanoma: 10–15% 5-year survival.

HIGH-YIELD FACTS IN

The Appendix

Embryology and Development 168

Anatomy/Structure 168

Acute Appendicitis 169

- Incidence 169
- Pathophysiology 169
- Symptoms 169
- Signs 169
- differential diagnosis 170
- diagnosis 170
- treatment 170

Appendicitis in Special Populations 173

- Pregnant Patients 173
- elderly Patients 173
- Immunocompromised Patients (e.g., aids Patients, recipients of high-dose chemotherapy) 173

Appendiceal Neoplasms 173

- carcinoid 173
- mucinous tumors 174
- adenocarcinoma 174

Appendiceal Abscess 174

Recent Advances 174

Embryology and Development

- The appendix begins to bud off from the cecum at around the **sixth week** of embryological development.
- The base of the appendix remains in a fixed position with respect to the cecum, whereas the tip can end up in various positions (Figure 11-1).

Anatomy/Structure

- **Mesoappendix** is the mesentery that suspends the appendix from the terminal ileum. It contains the **appendicular artery,** the blood supply to the appendix.
  - The appendicular artery is a branch of the ileocolic artery.
  - The ileocolic artery is a branch of the superior mesenteric artery.
- The appendix is composed of the same layers as the colon wall.
  - Mucosa, submucosa, inner circular muscle, outer longitudinal muscle, serosa.
  - The three distinct bands of outer longitudinal muscle, the **taeniae coli,** converge on the appendix.
  - Although many have claimed that the appendix is merely a vestigial organ, it is actually an immunological organ and **secretes IgA.** However, it is not an essential organ and can be removed without immunological compromise.
- The length can range from 2 to 20 cm but averages **6–9 cm.**
- Luminal capacity is < 1 mL.

OR TIP

After locating the cecum, one can easily find the appendix by following the three taeniae coli until they converge at the base of the appendix.

EXAM TIP

The lifetime incidence of acute appendicitis in the United States is about 7%.

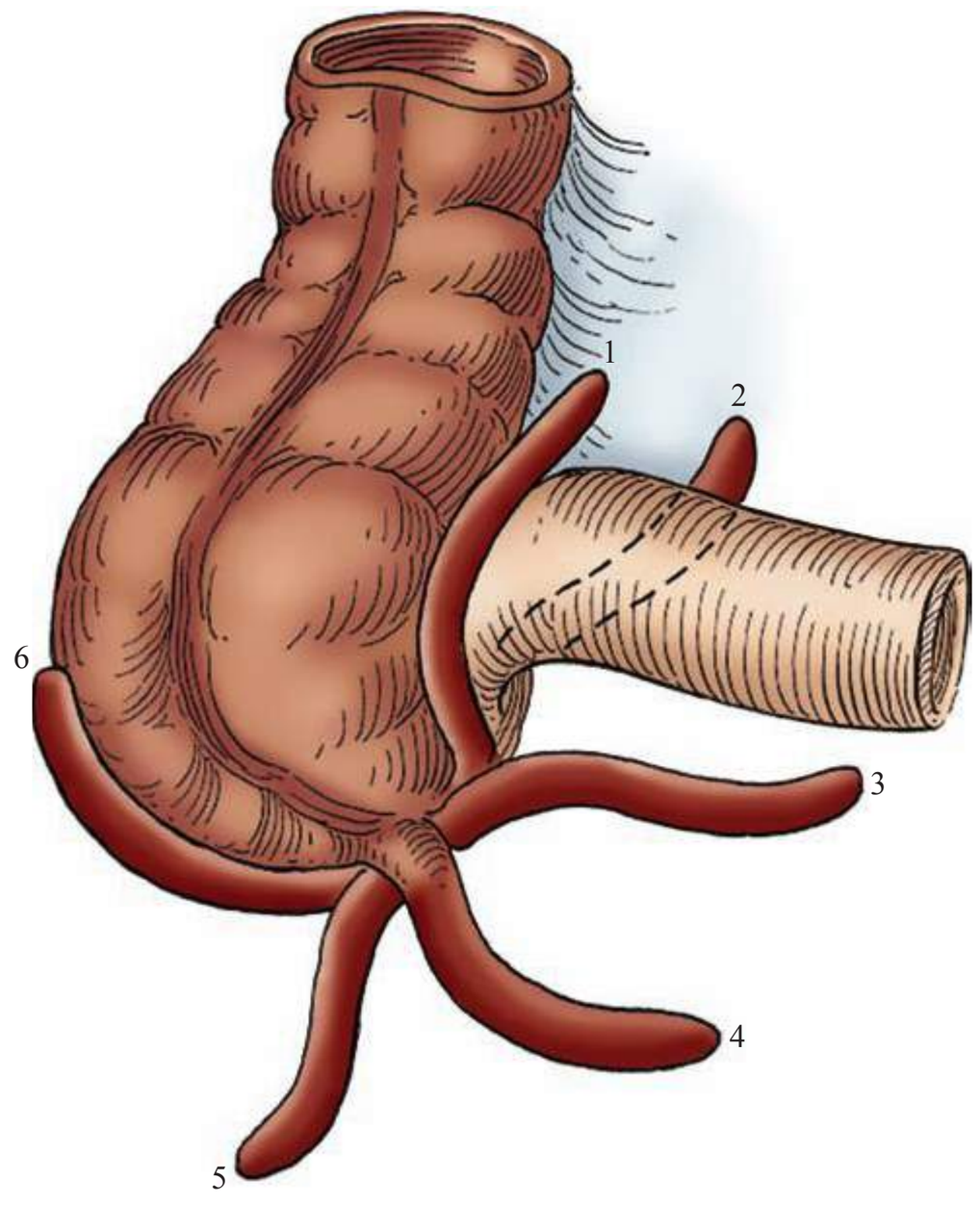


FiGURe 11-1. Anatomic variation in the position of the appendix. (1) Preileal; (2) postileal; (3) promontoric; (4) pelvic; (5) subcecal; (6) paracolic or prececal. (Reproduced from Smink DS, Soybel DI. Appendix and appendectomy. In Zinner MJ, Ashley SW, eds. Maingot's Abdominal Operations. 11th ed. New York: McGraw-Hill; 2007: 590.)

Acute Appendicitis

Incidence

One of the most common acute surgical diseases.

- Highest in early adulthood, at the peak of lymphoid tissue growth.
- Second peak in the incidence of appendicitis occurs in the elderly.
- There is a higher incidence of appendicitis in males than females (1.3:1).

Pathophysiology

The probable sequence of events in acute appendicitis is:

1. Luminal **obstruction.**
   - In young patients, more commonly by **lymphoid tissue hyperplasia.**
   - In older patients, **fecalith** is an increasingly common cause of obstruction.
2. **Distention** and increased intraluminal pressure.
   - The appendiceal mucosa continues to secrete normally despite being obstructed.
   - The resident bacteria multiply rapidly, further increasing intraluminal pressure.
3. Venous congestion.
   - The intraluminal pressure eventually exceeds capillary and venule pressures.
   - Arteriolar blood continues to flow in, causing vascular congestion and engorgement.
4. Impaired blood supply renders the mucosa ischemic and susceptible to **bacterial invasion.**
5. **Inflammation** and **ischemia** progress to involve the serosal surface of the appendix.

Symptoms

- Right lower quadrant (RLQ) pain.
- **Migration** of pain from the periumbilical region to localize in the RLQ.
- Nausea/vomiting.
- Fever.

Signs

- Direct rebound tenderness, which is maximal at (or around) McBurney's point.
- Leukocytosis.
- Rovsing's sign: Palpation pressure exerted over left lower quadrant (LLQ) causes pain in RLQ.
- Iliopsoas sign: Pain on extension at the right hip.
  - The patient will get relief by flexing the right thigh at the hip, which relaxes the psoas muscle.
  - This sign signifies retrocecal appendicitis.
- Obturator sign (signifies pelvic appendicitis): Pelvic pain on internal rotation of the right thigh.

WARD TIP

The initial dull, diffuse (visceral) pain that occurs at the onset of acute appendicitis is a result of the stimulation of visceral afferent stretch fibers. These nerve endings fire as a result of the sudden-onset distention, and the pain is commonly felt around the umbilicus (T10 distribution).

EXAM TIP

If clinical presentation is most likely appendicitis (umbilical pain migrating to RLQ, fever, nausea and vomiting, elevated WBC) go straight to appendectomy. Imaging is not necessary.

WARD TIP

The shift from dull, diffuse (visceral) pain to sharp, localized (somatic) RLQ pain occurs when the inflamed serosa contacts the parietal peritoneum, causing peritoneal irritation. This pain is felt in the area directly overlying the appendix.

EXAM TIP

McBurney's point: One third the distance along a line drawn from the right anterior superior iliac spine to the umbilicus.

EXAM TIP

Yersinia enterolytica can cause mesenteric adenitis and is a great mimicker of appendicitis.

Differential Diagnosis

Gastrointestinal Conditions

- Gastroenteritis
- Mesenteric adenitis
- Meckel's diverticulum
- Intussusception
- Typhoid fever
- Primary peritonitis

Genitourinary Conditions

- Ectopic pregnancy
- Pelvic inflammatory disease
- Ovarian torsion/cyst/tumor
- Urinary tract infection/pyelonephritis
- Ureteral stone

Diagnosis

WARD TIP

The appendix becomes compressible after perforation, and therefore will lead to a false negative ultrasound (a "noncompressible" appendix on ultrasound is diagnostic for a non-perforated appendicitis).

Labs

- **Complete blood count:**
  - Leukocytosis (> 10,000 in 90% of cases), usually with concomitant left shift (polymorphonuclear neutrophil [PMN] predominance).
  - Consider perforation or abscess if WBC > 18,000.
- **Urinalysis:**
  - Helpful in ruling out genitourinary causes of symptoms.
  - RBCs and WBCs may be present secondary to extension of appendiceal inflammation to the ureter.
  - Significant hematuria or pyuria, and bacteriuria from a catheterized specimen should suggest underlying urinary tract pathology.
- **Abdominal x-ray:**
  - Diagnostic Imaging
  - Not particularly useful in most cases.
  - May reveal appendicolith/fecalith (< 15% of cases).
- **Abdominal CT with contrast:**
  - Very sensitive (95–98%) and somewhat specific (83–90%).
  - Useful in identifying several other inflammatory processes that may present similarly to appendicitis.
  - Positive findings include:
    - Dilatation of appendix to > 6 mm in diameter.
    - Thickening of appendiceal wall (representing edema).
    - Periappendiceal streaking (densities within perimesenteric fat).
    - Presence of appendicolith (see Figure 11-2).
- **Graded compression ultrasonography:**
  - Sensitivity of 85% and specificity of 92% for diagnosing appendicitis.
  - Positive finding: Enlarged (> 6 mm), noncompressible appendix.
  - Especially useful in ruling out gynecologic pathology.
    - Useful in pregnancy.
    - MRI is used in pregnancy instead of CT scan.

Treatment

See Figure 11-3.

- Preoperative: IV fluids plus antibiotics.
- Prompt appendectomy (open or laparoscopic) is recommended.
  - Laparoscopic approach is preferred. It is associated with reduction in surgical site infection, shorter hospital stay, and reduction in long-term risk of bowel obstruction.

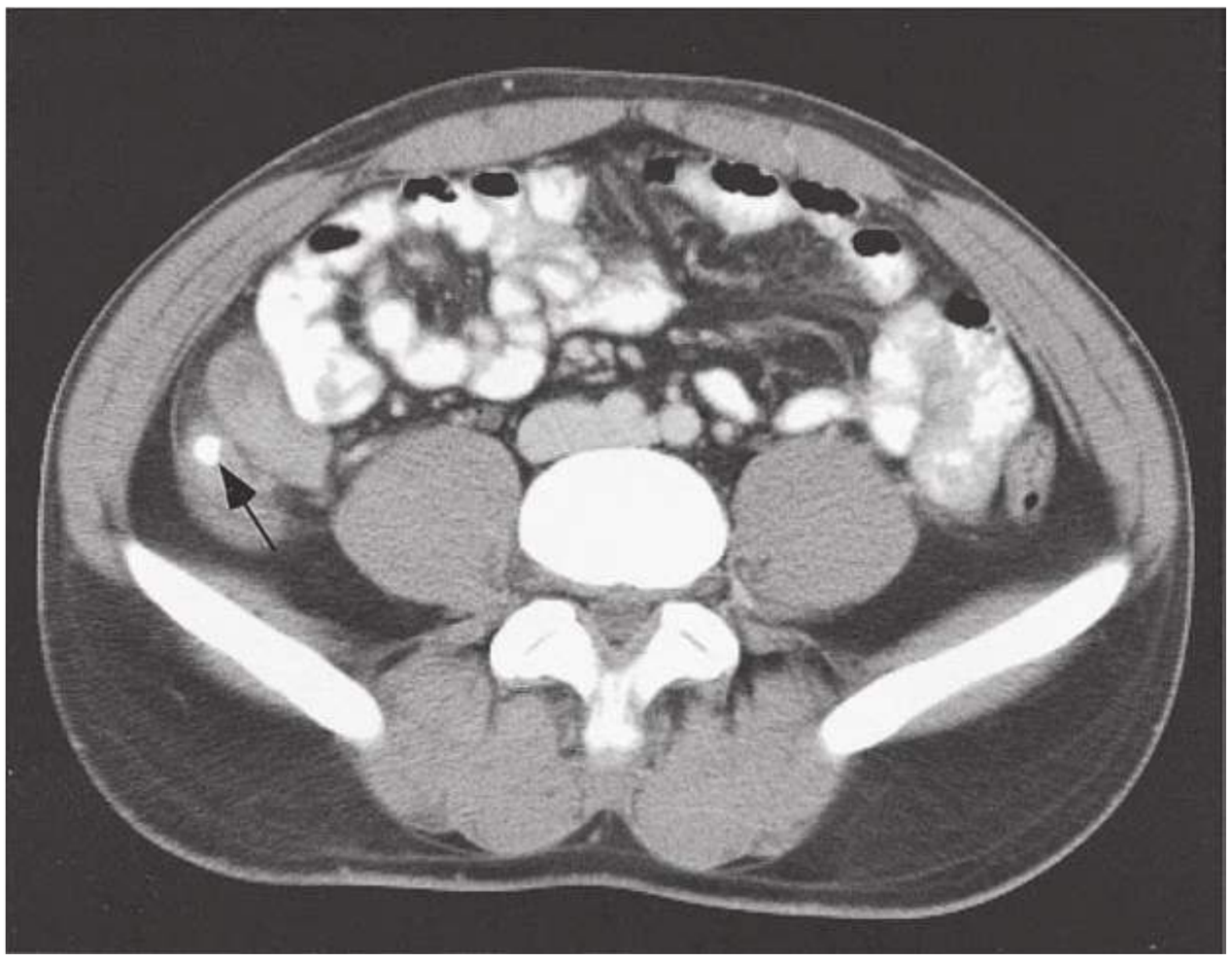


FIGURE 11-2. Abdominal CT scan demonstrating appendicolith and acute appendicitis.

- For free perforation: Peritoneal washout and parenteral antibiotics.
  - For localized perforation (with abscess) CT scan guided drainage and interval appendectomy. (An "interval appendectomy" is an elective appendectomy performed after successful medical management of appendicitis to prevent recurrence. Its necessity remains controversial.)
  - If greater than 5 days of onset of symptoms, or if abscess is present, one can manage medically with antibiotics. No appendectomy is done unless there is failure to respond to antibiotics or appendicitis recurs.

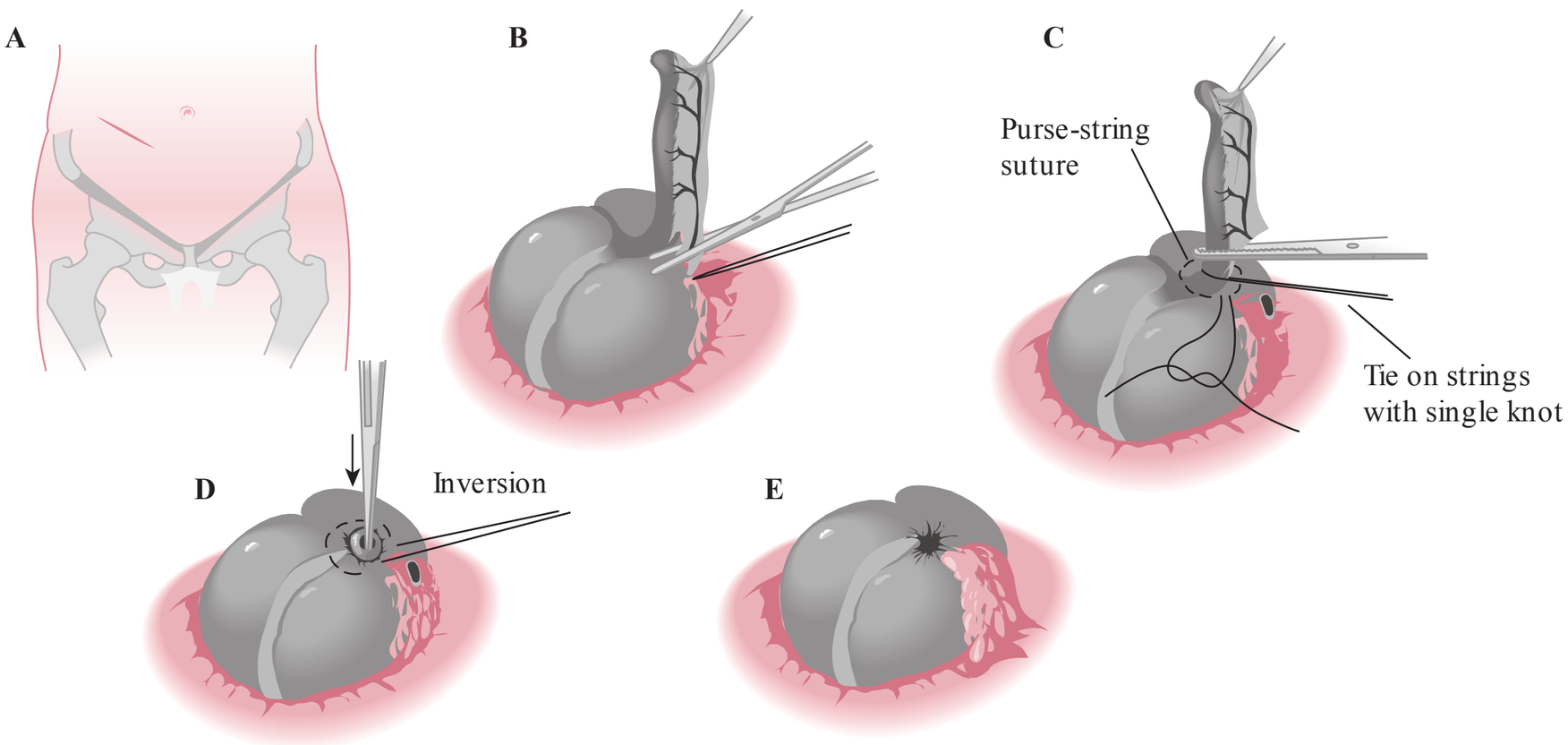
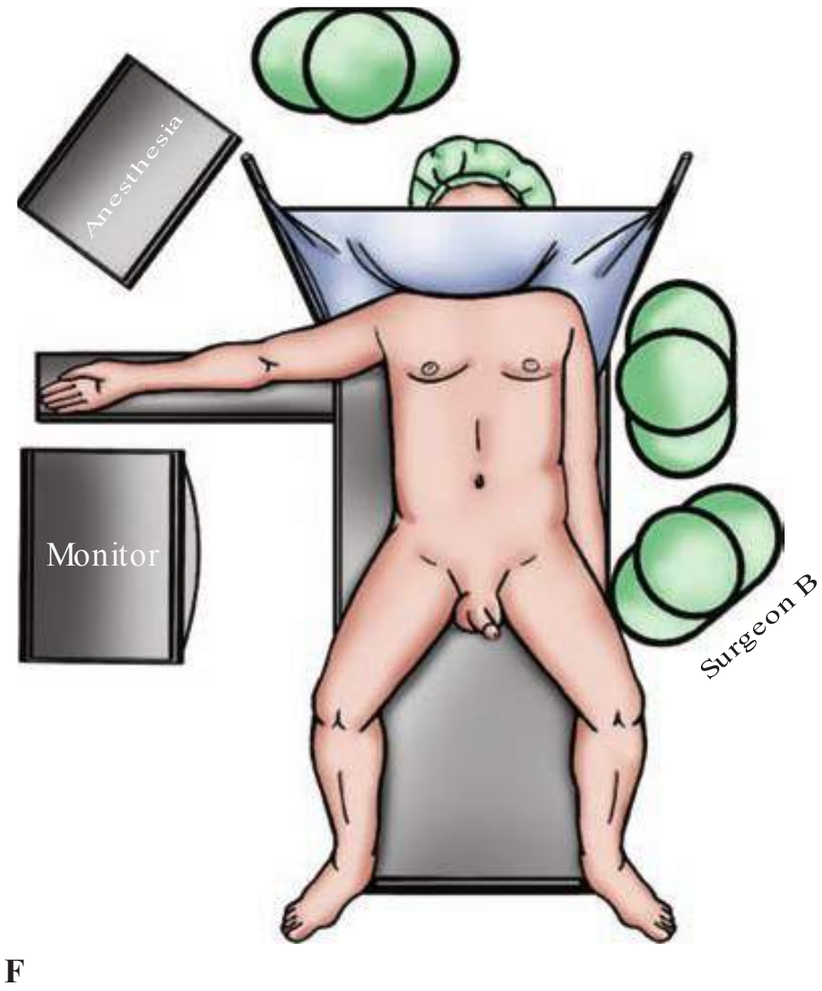
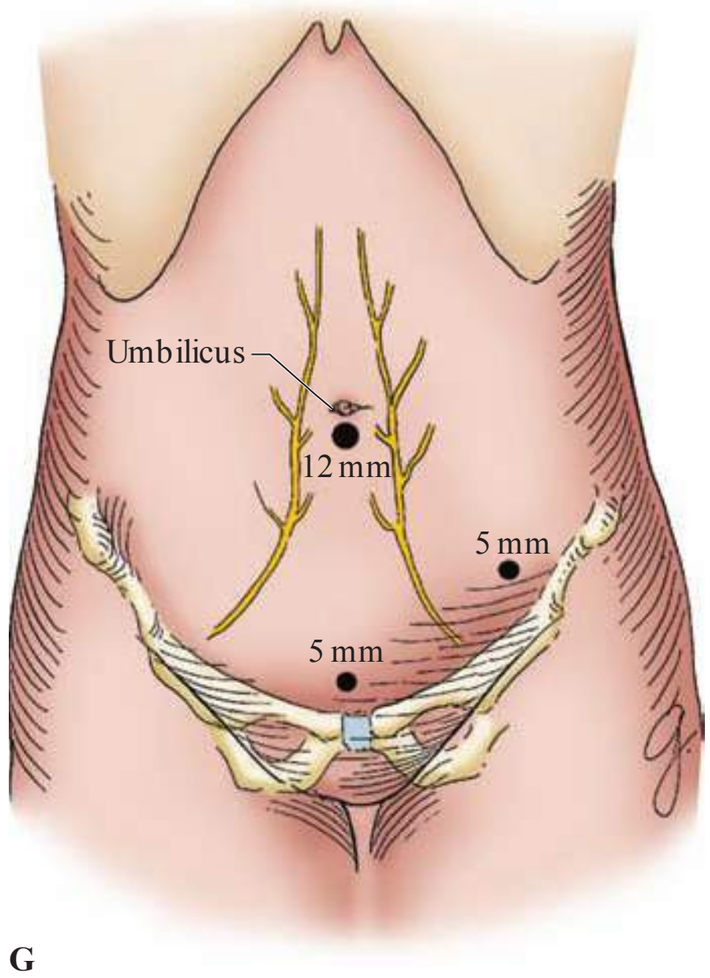


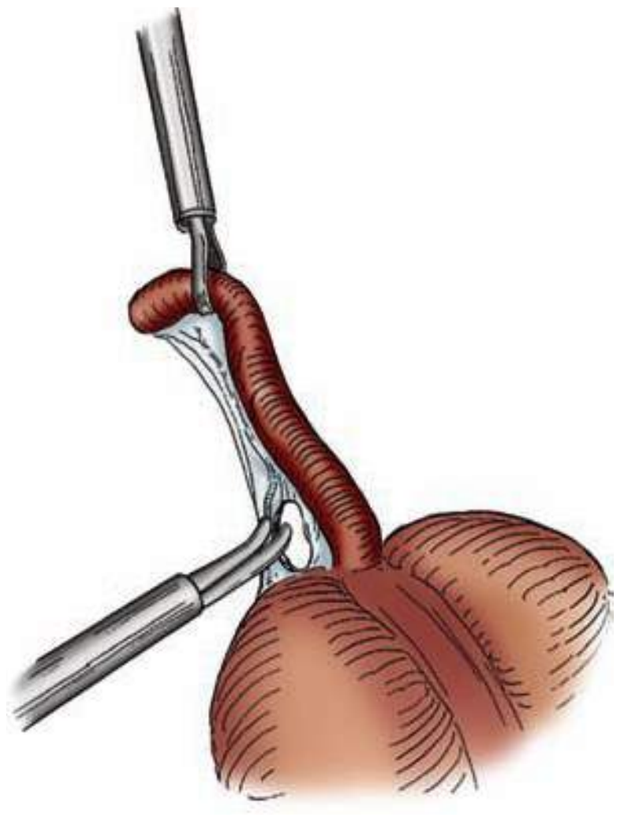
FIGURE 11-3 A-K. Technique of open appendectomy. A: Incision. B: After delivery of the tip of the cecum, the mesoappendix is divided. C: The base is clamped and ligated with a simple throw of the knot. The next step—inversion of the stump—is optional. D: A clamp is placed to hold the knot during inversion with a purse-string suture of fine silk. E: The loosely tied inner knot on the stump assures that there is no closed space for the development of a stump abscess. (A-E: Reproduced, with permission, from Doherty GM. Current Diagnosis & Treatment: Surgery. 14th ed. New York, NY: McGraw-Hill Education; 2015. Figure 28-1.) (Continued)



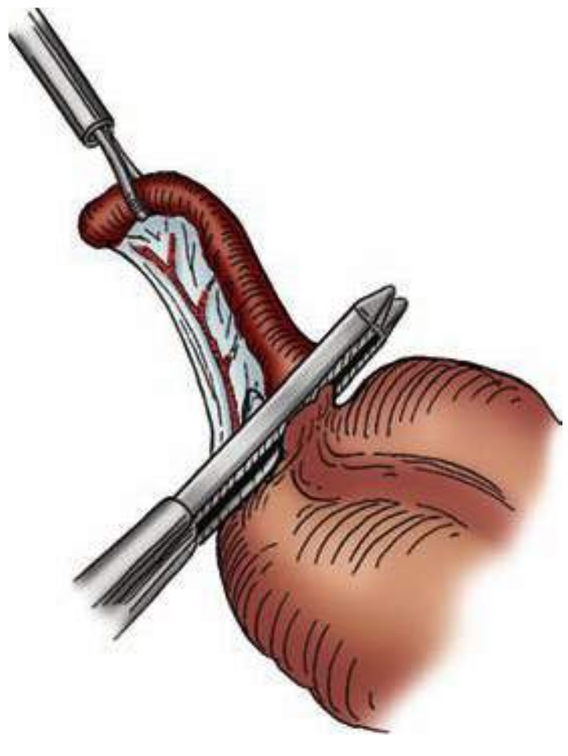
F



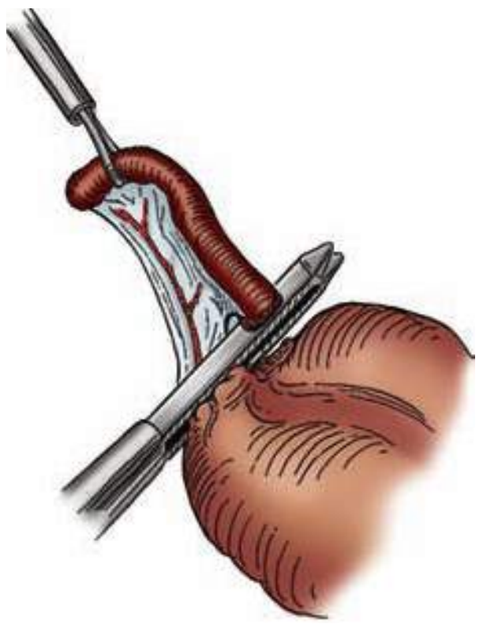
G



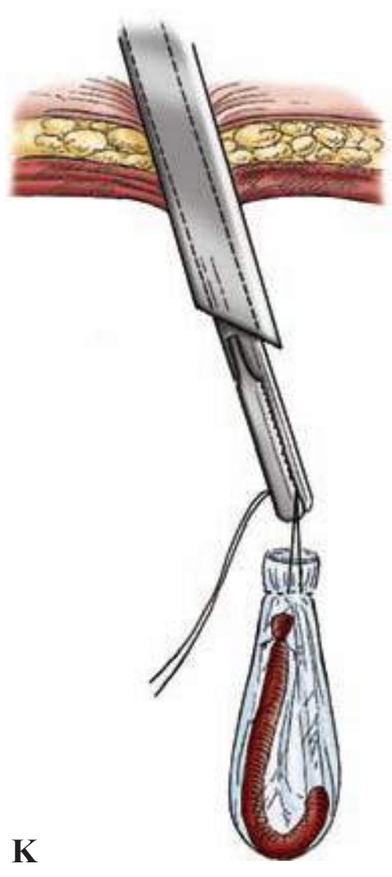
H



I



J



K

FiGURe 11-3A-K. F-K: Laparoscopic appendectomy technique. (F-K: Reproduced, with permission, from Zinner MJ, Ashley SW. Maingot's Abdominal Operations. 12th ed. New York, NY: McGraw-Hill Education; 2013. Figure 31-8.)

Appendicitis in Special Populations

Pregnant Patients

- Appendicitis is the most common surgical emergency in pregnant patients.
- Fetal mortality increases 3–8% with appendicitis and 30% with perforation.
- **Surgery** is the standard treatment, though 10–15% of women will experience premature labor.

WARD TIP

The enlarged uterus may push the appendix up, explaining why pregnant patients may present with RUQ pain.

elderly Patients

- Tend to present atypically, leading to delayed diagnosis.
  - Present later in the course and with less pain, **may present as a small bowel obstruction.**
  - Delayed leukocytosis.
- Higher risk of perforation and higher mortality than in younger patients.

Immunocompromised Patients (e.g., AIDS Patients, recipients of high-dose chemotherapy)

- Although they may not have absolute leukocytosis, compared to baseline WBC count, they will demonstrate relative leukocytosis.
- The differential diagnosis is expanded to include opportunistic infections such as cytomegalovirus (CMV)-related bowel perforation and neutropenic colitis.

EXAM TIP

Patients will not typically have carcinoid syndrome unless the tumor has metastasized to the liver.

Appendiceal Neoplasms

carcinoid

A relatively low-grade neuroendocrine tumor (it secretes enzymes aberrantly; the enzymes typically cause nausea, diarrhea, and flushing).

- The appendix is the most common site of carcinoid tumors in the GI tract.
- Carcinoid is the second most common type of appendiceal tumor (commonest being mucinous adenocarcinoma).

diagnosis

Increased urinary 5-hydroxyindoleacetic acid (5-HIAA) and increased serum serotonin.

Treatment

Size is the major determinant of treatment and malignant potential:

- Tumors < 2 cm are treated with appendectomy.
- Tumors > 2 cm or at base of the appendix, are treated with right hemicolectomy.
- Serotonin antagonists (e.g., cyproheptadine) or somatostatin analogues (e.g., octreotide) can be used for symptoms of carcinoid syndrome.

EXAM TIP

Patients with carcinoid syndrome should also be monitored for pellagra/vitamin B_3/niacin deficiency (3 Ds—dementia, dermatitis, and diarrhea), because tryptophan is used up to make serotonin, instead of niacin.

mucInous tumors

- Can rupture, causing **pseudomyxoma peritonei** with mucin implants on peritoneal surfaces and the omentum.
  - More common in women (ratio of 3:2).
  - Complications include bowel obstruction and perforation.
- Have been associated with migratory thrombophlebitis.

adenocarcInoma

- Colon cancer that arises from the appendix; very rare and almost never diagnosed preoperatively.
- Rapid spread to regional lymph nodes, ovaries, and peritoneal surfaces.
- If confined to appendix and local lymph nodes, right hemicolectomy is the treatment of choice.

EXAM TIP

Think of an abscess in a patient who presents with the clinical picture of **appendicitis and RLQ mass.**

Appendiceal Abscess

- **Signs and symptoms:** Similar to acute appendicitis.
  - Increasing RLQ pain.
  - Tender, fluctuant RLQ mass that is palpable on rectal examination.
  - Anorexia.
  - Fever.
  - Localizing peritonitis.
  - Leukocytosis.
- **Diagnosis:** Confirmed by CT scan.
- **Treatment:** Percutaneous or operative drainage.

Recent Advances

Natural orifice transluminal endoscopic surgery (NOTES): In this procedure the appendix is removed via upper gastrointestinal endoscopy with the surgeon operating through the gastric wall and ultimately removing the appendix through the mouth without any external scar. The gastrotomy is closed from within the stomach.

It can also be done via the vagina in females.

NOTES is only being performed in limited centers worldwide under clinical study protocols.

HIGH-YIELD FACTS IN

Hernia and abdominal Wall Problems

Overview 176

Definitions 176

Classification of Hernias 176

General Groin Anatomy 176

- Layers of the abdominal Wall 176
- internal Structures 176
- nerves 177
- femoral canal Structures 177
- anatomical triangles 177

Inguinal Hernia 178

- risk factors 178
- Symptoms 178
- diagnosis 178
- differential diagnosis 178
- radiology 178
- management 179

Types of Groin Hernia Surgery 180

- open 180
- Laparoscopic 180
- indications for Surgery 181
- complications of groin hernia Surgery 181
- prognosis 181

Classification of Inguinal Hernias 181

- direct inguinal hernia 181
- indirect inguinal hernia 181

Femoral Hernia 182

- anatomy 182
- Symptoms 182
- differential diagnosis 182

Acquired Umbilical Hernia 182

- associated factors 182
- complications 183
- treatment 183

Pediatric Umbilical Hernia 183

- management 183

Pediatric Wall Defects 183

Esophageal Hiatal Hernia 183

- sliding esophageal hernia (type i) 184
- paraesophageal hiatal hernia (type ii) 184

Other Hernias 184

EXAM TIP

The most common hernia in males and females is the indirect inguinal hernia.

WARD TIP

Groin hernias usually present with the complaint of a bulge in the inguinal region. The patient may describe minor pain or vague discomfort.

Overview

- Hernias are a common surgical problem.
- It is estimated that 10% of the population develops some types of hernia during life and that they are present in 3–4% of the male population.
- Fifty percent are indirect inguinal hernias, 25% are direct inguinal, and 15% are femoral.
- The male-to-female ratio is 7:1.
- Abdominal wall hernias are the most common condition requiring major surgery.

Definitions

- **Hernia:** A protrusion of a viscus through an abnormal opening in the wall of a cavity in which it is contained.

Classification of Hernias

- **External hernia:** The sac protrudes completely through the abdominal wall. Examples: Inguinal (indirect and direct), femoral, umbilical, and epigastric.
- **Intraparietal hernia:** The sac is contained within the abdominal wall. Example: Spigelian hernia.
- **Internal hernia:** The sac is within the visceral cavity. Examples: Diaphragmatic hernias (congenital or acquired) and the small intestine herniating in the paraduodenal pouch.
- **Reducible hernia:** The protruding viscus can be returned to the abdomen.
- **Irreducible (incarcerated) hernia:** The protruding viscus cannot be returned to the abdomen.
- **Strangulated hernia:** The vascularity of the viscus is compromised—**surgical emergency.**

General Groin Anatomy

See Figure 12-1.

Layers of the abdominal wall

Skin, subcutaneous fat, Scarpa's fascia, external oblique muscle, internal oblique muscle, transversus abdominis muscle, transversalis fascia, peritoneal fat, and peritoneum.

internal structures

- Inguinal canal: Length, 4 cm; boundaries:
  - Anterior wall: External oblique aponeurosis.
  - Posterior wall: Transverse abdominal muscle aponeurosis and transversalis fascia.
- Spermatic cord: Begins at the deep ring and contains the vas deferens and its artery (descend to the seminiferous tubules), one testicular artery and two to three veins, lymphatics (incline superiorly to the kidney region), autonomic nerves, and fat.

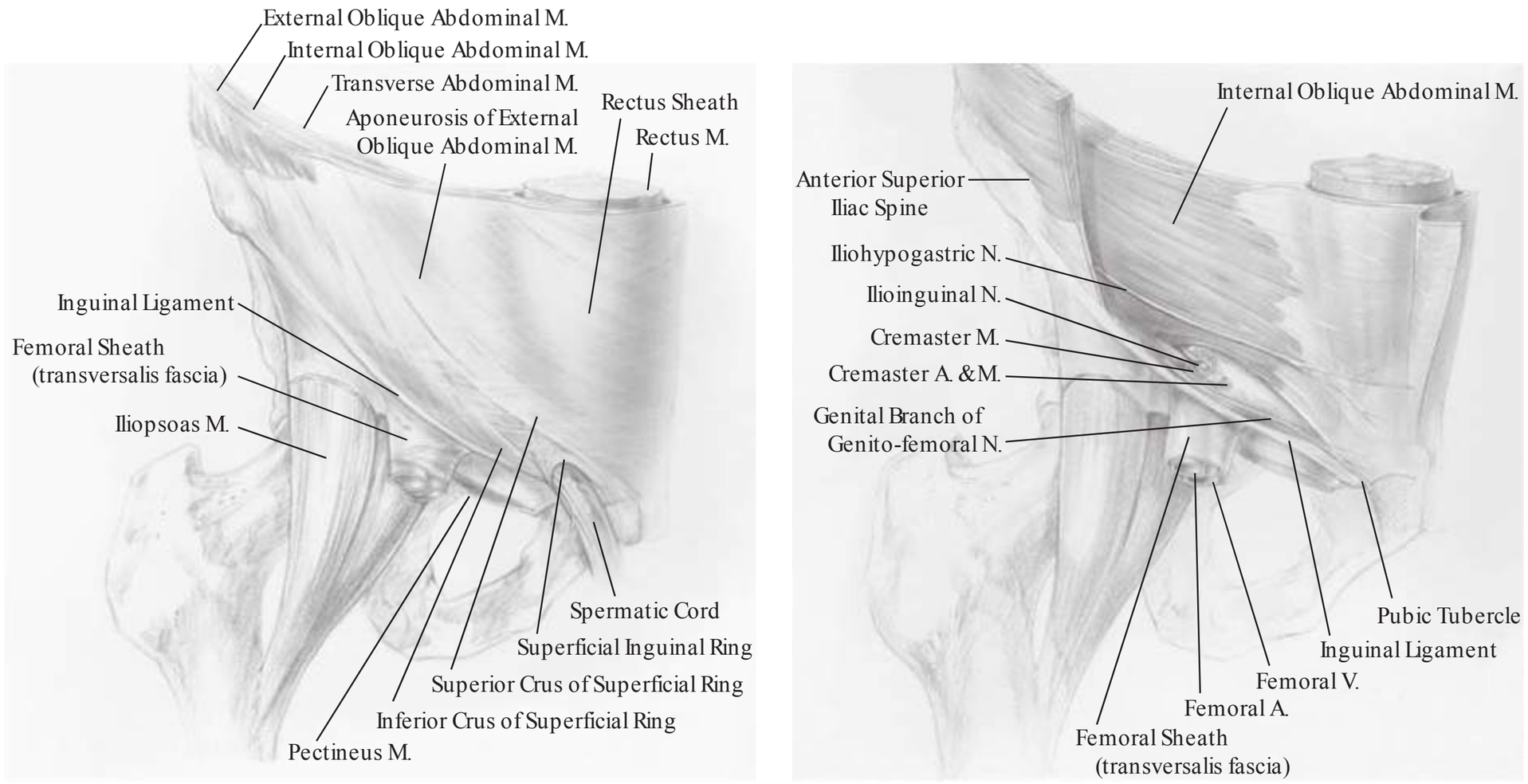


FiGUre 12-1. Groin anatomy.

nerves

- Genital:
  - Location: Travels along with the cremaster vessels to form a neurovascular bundle.
  - Originates: From L1 and L2.
  - Motor and sensory: Innervates the cremaster muscle, skin of the side of the scrotum, and labia.
  - May substitute for the ilioinguinal nerve if it's deficient.
- Iliohypogastric, ilioinguinal nerves, and the genital branch of the genitofemoral nerve:
  - Iliohypogastric and ilioinguinal intertwine.
  - Originates: From T12 and L1.
  - Sensory: To skin of groin, base of penis, and medial upper thigh.
- Genital branch of genitofemoral nerve: Located on top of the spermatic cord in 60% of people but can be found behind or within the cremaster muscle. Often cannot be found or is too small to be seen.

femoral canal structures

From lateral to medial: **N**erve, **A**rtery, **V**ein, **E**mpty space, **L**ymph nodes.

anatomical triangles

- **Hesselbach's triangle:** The triangular area in the lower abdominal wall. It is the site of direct inguinal hernia. The boundaries of Hesselbach's triangle are:
  - Inferior border: Inguinal ligament.
  - Medial border: Rectus abdominis.
  - Lateral border: Inferior epigastric vessels (lateral umbilical fold).
- **Triangle of Grynfeltt** (superior lumbar) bounded by the 12th rib superiorly and the internal oblique muscle anteriorly, with the floor composed of fibers of the quadratus lumborum muscle.

WARD TIP

The inguinal area is examined with the patient standing and facing the physician. Presence of a discrete bulge in the inguinal area reveals the hernia. Valsalva's maneuver and cough may accentuate the bulge, making it clearly visible or palpable.

WARD TIP

Memory aid for the femoral canal structures: **n a Ve l .**

EXAM TIP

md 's l ie: Medial to inferior epigastric vessels in Direct hernia, Lateral to inferior epigastric vessels in Indirect hernias.

- **Triangle of Petit** (inferior lumbar triangle) bounded by:
  - Posteriorly: Latissimus dorsi muscle.
  - Anteriorly: External oblique muscle.
  - Inferiorly: Iliac crest.
  - The floor is composed of fibers from the internal oblique and transversus abdominis muscle.

Inguinal Hernia

- Hernias arising above the abdominocrural crease.
- Most common site for abdominal hernias.
- Male-to-female ratio: 25:1.
- Males: Indirect > direct (2:1).
- Female: Direct is rare.
- Incidence, strangulation, and hospitalization all increase with age.
- Cause 15–20% of intestinal obstructions.

risk factors

- Abdominal wall hernias occur in areas where aponeurosis and fascia are devoid of protecting support of striated muscle.
- They can be congenital or acquired by surgery or muscular atrophy.
- Female predisposition to femoral hernias: Increased diameter of the true pelvis as compared to men, proportionally widens the femoral canal.
- Muscle deficiency of the internal oblique muscles in the groin exposes the deep ring and floor of the inguinal canal, which are further weakened by intra-abdominal pressure.
- Connective tissue destruction (transverse aponeurosis and fascia): Caused by physical stress secondary to intra-abdominal pressure; smoking; aging; connective tissue disease; systemic illnesses; fracture of elastic fibers; alterations in structure, quantity, and metabolism of collagen.
- Other factors: Abdominal distention, ascites with chronic increase in intra-abdominal pressure, and peritoneal dialysis.

EXAM TIP

Patent processus vaginalis are causes for both indirect inguinal hernias and for hydroceles.

symptoms

- Asymptomatic: Some patients have no symptoms.
- Symptomatic: Wide variety of nonspecific discomforts related to the contents of the sac and the pressure by the sac on adjacent tissue.
- Pain: Worse at the end of the day and relieved at night when patient lies down (because the hernia reduces).
- Groin hernias do not usually cause testicular pain. Likewise, testicular pain doesn't usually indicate the onset of a hernia.

WARD TIP

Hydroceles can resemble an irreducible groin hernia. To distinguish, transilluminate (hernias will not light up).

diagnosis

Physical exam: In the standing position, have patient strain or cough. The hernia sac with its contents will enlarge and transmit a palpable impulse.

differentiaL diagnosis

See Table 12-1.

radioLogy

Hernia is a clinical diagnosis, and radiological tests (ultrasound/CT scan) are done only in special circumstances (e.g., morbid obesity, where clinical examination is not reliable).

Table 12-1. Differential Diagnosis of Groin Hernia

Malignancy
Lymphoma
Retroperitoneal sarcoma
Metastasis
Testicular tumor
Primary testicular
Varicocele
Epididymitis
Testicular torsion
Hydrocele
Ectopic testicle
Undescended testicle
Femoral artery aneurysm or pseudoaneurysm
Lymph node
Sebaceous cyst
Hidradenitis
Cyst of the canal of Nuck (female)
Saphenous varix
Psoas abscess
Hematoma
Ascites

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York: McGraw-Hill Education; 2015. Table 37-5.

management

Principles of Treatment

- Tension-free repair of the hernia defect.
- Repair using fascia, aponeurosis, or mesh.
- Suture material used should hold until fibrous tissue is formed over it.
- Resuscitation in case of strangulated hernia with gangrene, with shock, or with intestinal obstruction.

nonsurgical

- No role of medical management in a patient who can tolerate surgery.
- Can be considered in moribund patients.
- Hernia truss is a device to keep a reducible hernia contained by external pressure. Its use is very limited in modern practice.

surgical

- Treatment modality of choice.
- Herniotomy is the operation where hernia sac is identified, freed, its neck ligated, and the sac reduced. This may be sufficient in young, muscular individuals and in children.
- Herniorrhaphy and hernioplasty are herniotomy along with repair of the posterior wall of the inguinal canal and the internal ring.

Types of Groin Hernia Surgery

open

- **Tissue repair:** Uses the patient's tissue for reinforcement.
  - Bassini's repair: Suturing of conjoint tendon to the incurved part of inguinal ligament. Bassini's repair is still widely practiced as it is simple to perform with good results.
  - Shouldice repair: Double breasting of transversalis fascia. Technically challenging with excellent results.
  - McVay's repair/Cooper's repair (used for femoral hernias): Conjoint tendon sutured to Cooper's ligament.
- **Mesh repair:** Uses prosthetic mesh and/or plug for tension-free repair (preferred).
  - Anterior approach: For example, Lichtenstein's repair (most common).
  - Posterior approach: For example, Kugel's method, Stoppa's repair.

Laparoscopic

- Requires an experienced laparoscopic surgeon, has decreased postop pain, requires general or regional anesthesia, and more expensive. Wound infection has been shown to decrease with laparoscopic repair.
  - Transabdominal pre-peritoneal procedure (TAPP).
  - Totally extraperitoneal procedure (TEP)—preferred.
- The choice of hernia surgery depends on the surgeon's expertise. TEP is being performed in larger numbers as surgical proficiency in laparoscopy increases, and the results are promising (see Figure 12-2).

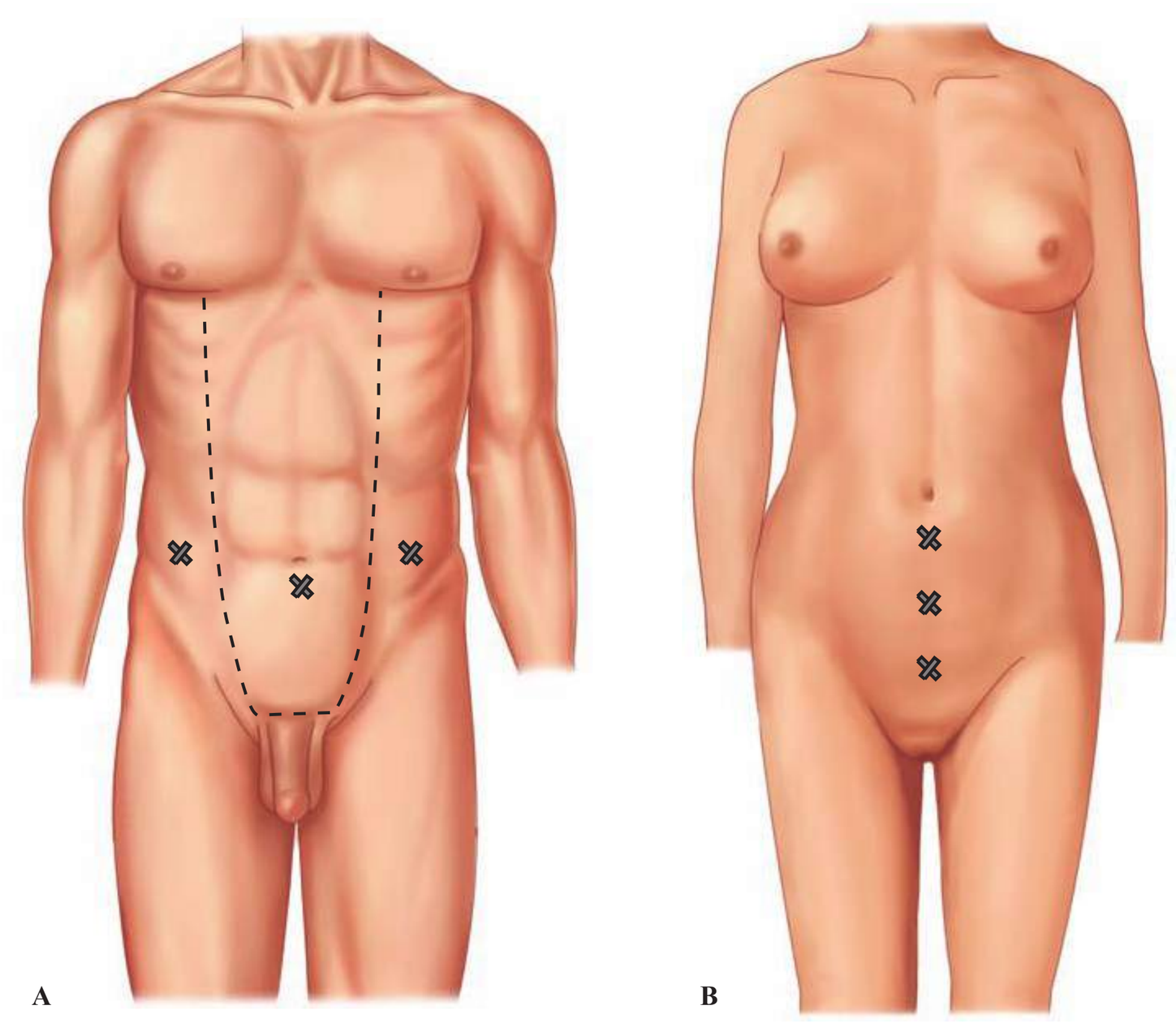


FiGUre 12-2. Trocar placement for (a) transabdominal preperitoneal repair and (b) totally extraperitoneal repair. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Figure 37-24.)

indications for surgery

Generally, all hernias should be repaired unless the risks of surgery outweigh the benefits of the repair.

complications of groin hernia surgery

- Chronic groin pain.
  - Cord and testicular (hematoma, injury of vas deferens, ischemic orchitis, testicular atrophy).
  - Mesh-related complications (infection, erosion, displacement, contraction, fracture).
  - Laparoscopic and general complications.

prognosis

Recurrence:

- Recurrence rates of about 1% are seen with the Shouldice repair and as low as 0.2% with the tension-free mesh repair.
- Caused by excessive tension on repair, deficient tissue, inadequate hernioplasty, or overlooked hernias.
- More common with direct hernias.

Classification of Inguinal Hernias

- Direct
- Indirect

direct inguinal hernia

A direct inguinal hernia enters the inguinal canal through its weakened posterior wall. The hernia does not pass through the internal ring.

- Lies posterior to the spermatic cord.
- Practically never enters the scrotum.
- Wide neck (strangulation uncommon).
- Occurs almost exclusively in males.
- Common in older age groups.
- Common in smokers due to weakened connective tissue.
- Predisposing factors: Hard labor, cough, straining, and so on.
- Can lead to damage to the ilioinguinal nerve.

symptoms

- Bulge in groin.
- Dull dragging pain in the inguinal region referred to testis.
- Pain increases with hard work and straining.

indirect inguinal hernia

Herniation through the internal inguinal ring traveling to the external ring. If complete, can enter the scrotum while exiting the external ring.

- If congenital, associated with a patent processus vaginalis.
- Bilateral in one third of cases.
- Most common hernia in both males and females.

WARD TIP

A reducible hernia in a patient with ascites should not be corrected until the ascites is controlled.

- Occurs at all ages.
- More common in males than in females.
- In the first decade of life, the right-sided hernia is more common than left (because of late descent of right testis).

Femoral Hernia

A form of indirect hernia arising out of the femoral canal beneath the inguinal ligament (medial to the femoral vessels).

- Female-to-male ratio of 2:1.
- Males affected are in a younger age group.
- Rare in children.
- Uncommon—around 2.5% of all groin hernias.
- Left side 1:2 right side: Secondary to the sigmoid colon tamponading the left femoral canal.
- Common in elderly patients.
- High incidence of incarceration due to narrow neck.
- Twenty-two percent strangulate after 3 months, and 45% after 21 months.

anatomy

- The femoral canal is 1.25 cm long and arises from the femoral ring to the saphenous opening.
- Femoral sac originates from the femoral canal through a defect on the medial side (common) or the anterior (uncommon) side of the femoral sheath.

symptoms

- Dull dragging pain in the groin, with swelling.
- If obstructed, can cause vomiting and constipation.
- If strangulated, can lead to severe pain and shock.
- Swelling arises from below the inguinal ligament.

differentiaL diagnosis

- Inguinal hernia.
- Saphenous varix.
- Enlarged femoral lymph node.
- Lipoma.
- Femoral artery aneurysm.
- Psoas abscess.

Acquired Umbilical Hernia

- Abdominal contents herniate through a defect in the umbilicus.
- Common site of herniation, especially in females.

associated factors

Ascites, obesity, and repeated pregnancies.

Complications

- Strangulation of the colon and omentum is common.
- Rupture occurs in chronic ascitic cirrhosis. Emergency portal decompression is needed.

Treatment

- Surgical repair:
  - < 2 cm fascial defect: Closed by loosely placed polypropylene suture.
  - > 2 cm fascial defect: Managed with a prosthesis repair.
  - Mayo hemioplasty is the classical repair (not used often).

Pediatric Umbilical Hernia

- Secondary to a fascial defect in the linea alba with protruding abdominal contents, covered by umbilical skin and subcutaneous tissue.
- Caused by a failure of timely closure of the umbilical ring, and leaves a central defect in the linea alba.
- Common in infants.
- Incarceration is rare and reduction is contraindicated.

Management

Usually close spontaneously within 3 years if the defect is < 1.0 cm. Surgical repair indicated if:

- The defect > 2 cm.
- Child is > 4–5 years of age.
- Protrusion is disfiguring and disturbing to the child and parents.

Pediatric Wall Defects

See Pediatric Surgery chapter.

Esophageal Hiatal Hernia

- A hernia in which an anatomical part (such as the stomach) protrudes through the esophageal hiatus of the diaphragm.
- Three types of esophageal hiatal hernia are identified:
  1. The sliding hernia, type I, characterized by an upward dislocation of the cardia in the posterior mediastinum.
  2. The rolling or paraesophageal hernia, type II, characterized by an upward dislocation of the gastric fundus alongside a normally positioned cardia.
  3. The combined sliding-rolling or mixed hernia, type III, characterized by an upward dislocation of both the cardia and the gastric fundus.

The end stage of type I and type II hernias occurs when the whole stomach migrates up into the chest by rotating 180º around its longitudinal axis, with the cardia and pylorus as fixed points. In this situation, the abnormality is usually referred to as an intrathoracic stomach.

Sliding Esophageal Hernia (Type I)

- The gastroesophageal junction and the stomach herniate into the thoracic cavity.
- These account for more than 90% of all hiatal hernias.
- Can lead to reflux and esophagitis that can predispose to Barrett's esophagus.
- Management can be done medically with antacids and head elevation.
- Only 15% require surgery, consisting of wrapping of the stomach fundus around the lower esophageal sphincter (Nissen fundoplication).
- See diagram in GERD section of Esophagus chapter.

Paraesophageal Hiatal Hernia (Type II)

- Herniation of the stomach into the thorax by way of the esophageal hiatus, without disruption of the gastroesophageal junction.
- Rare (< 5%).
- High frequency of complications (i.e., obstruction, strangulation, and hemorrhage).
  - **Warrants prompt surgical correction** via transthoracic or transabdominal approach.
  - Both laparoscopic and open approaches can be used.
  - Reduction, repair with or without fundoplication is done.

Other Hernias

- **Richter's hernia:** Only part of the intestine wall circumference is in the hernia. May strangulate without obstruction. Seen commonly in femoral and obturator hernias.
- **Littre's hernia:** The hernial sac contains Meckel's diverticulum. It may become inflamed.
- **Garengoff's hernia:** The hernial sac has the appendix. Importance is that it may form an inflamed hernia.
- **Pantaloon hernia:** A combination of a direct and an indirect inguinal hernia.
- **Maydl's hernia:** W type of intestinal loop herniates; may strangulate with the gangrenous part being inside the abdomen, or may be reduced into the abdomen without noticing the gangrenous part.
- **Spigelian hernia:** The sac passes through the spigelian or semilunaris fascia.
- **Sliding inguinal hernia:** Any hernia in which part of the sac is the wall of a viscus. On the right, the cecum, ascending colon, or appendix is commonly involved; on the left, the sigmoid colon is involved.
- **Cooper's hernia:** Hernia that involves the femoral canal and tracts to the labia majora in females and to the scrotum in males.
- **Lumbar hernia:** Divided into congenital, spontaneous, traumatic, and incisional. Can pass through the triangle of Grynfeltt, through the inferior lumbar triangle of Petit, or previous incision.
- **Perineal hernia:** Located through pelvic diaphragm, anterior (passes through labia majora—females only) or posterior (male: enters the ischiorectal fossa; female: close to the vagina) to the superficial transverse perineal muscle.
- **Incisional hernia:** Resulting as a surgical complication. These could enlarge beyond repair. Associated with obesity, diabetes, and infection.
- **Eventration:** Loss of integrity of the abdominal wall, reducing the intra-abdominal pressure, and resulting in external herniation of bowel.

HIGH-YIELD FACTS IN

The hepatobiliary System

Hepatic Anatomy 186

Diagnostic Imaging 187
- Ultrasonography 187
- Cholangiogram 187
- hepatobiliary iminodiaCetiC aCid (hida) sCan 187
- CompUted tomography (Ct) sCan 188
- endosCopiC retrograde CholangiopanCreatography (er Cp) 188
- er Cp with sphinCterotomy (papillotomy) 188
- perCUtaneoUs transhepatiC Cholangiography (pt Ca) 188

Bile 188

Jaundice 189
- definition 189
- Signs and Symptoms 189
- diagnosis 189
- treatment 189

Liver Injury 189
- initial treatment 189
- nonoperative management 190
- operative management 190

Nontraumatic Liver Conditions 191
- hepatiC abSCesses 191
- hepatiC Cysts 192
- benign liver tUmors 193
- malignant liver tUmors 194

Liver Failure 196
- Child–pUgh sCore 196

Portal Hypertension 197
- definition 197
- CaUses 197
- Signs and Symptoms 197
- portosystemiC Collaterals and their CliniCal manifestations 197

Morbidities Resulting from Liver Failure 198
- hepatiC enCephalopathy 198
- asCites 199
- esophageal VariCes 199
- CapUt medUsa 200

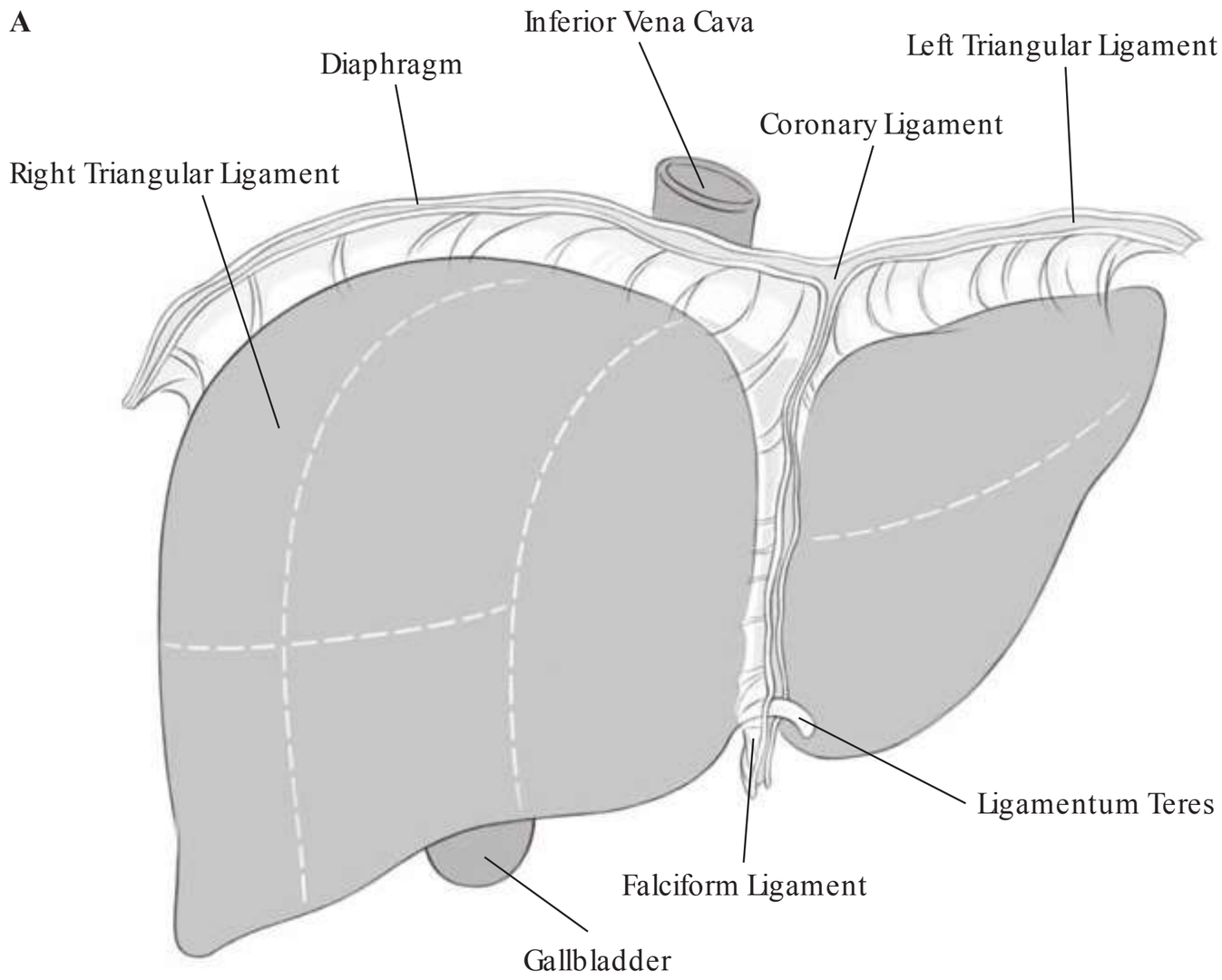
The Biliary System 200
- anatomy of the biliary tree 200
- anatomy of the gallbladder 200

Conditions of the Gallbladder and the Biliary Tree 201
- Cholelithiasis 201
- CholeCystitis 202
- CholedoCholithiasis 206
- Cholangitis 207
- gallstone ileUs 208
- CarCinoma of the gallbladder 209
- benign tUmors of the bile dUCts 210
- CholedoChal Cysts 210
- CholangioCarCinoma 211

Hepatic Anatomy

See Figure 13-1.

- Arterial supply:
  - Celiac trunk comes off the aorta, giving off the left gastric, splenic, and common hepatic arteries.



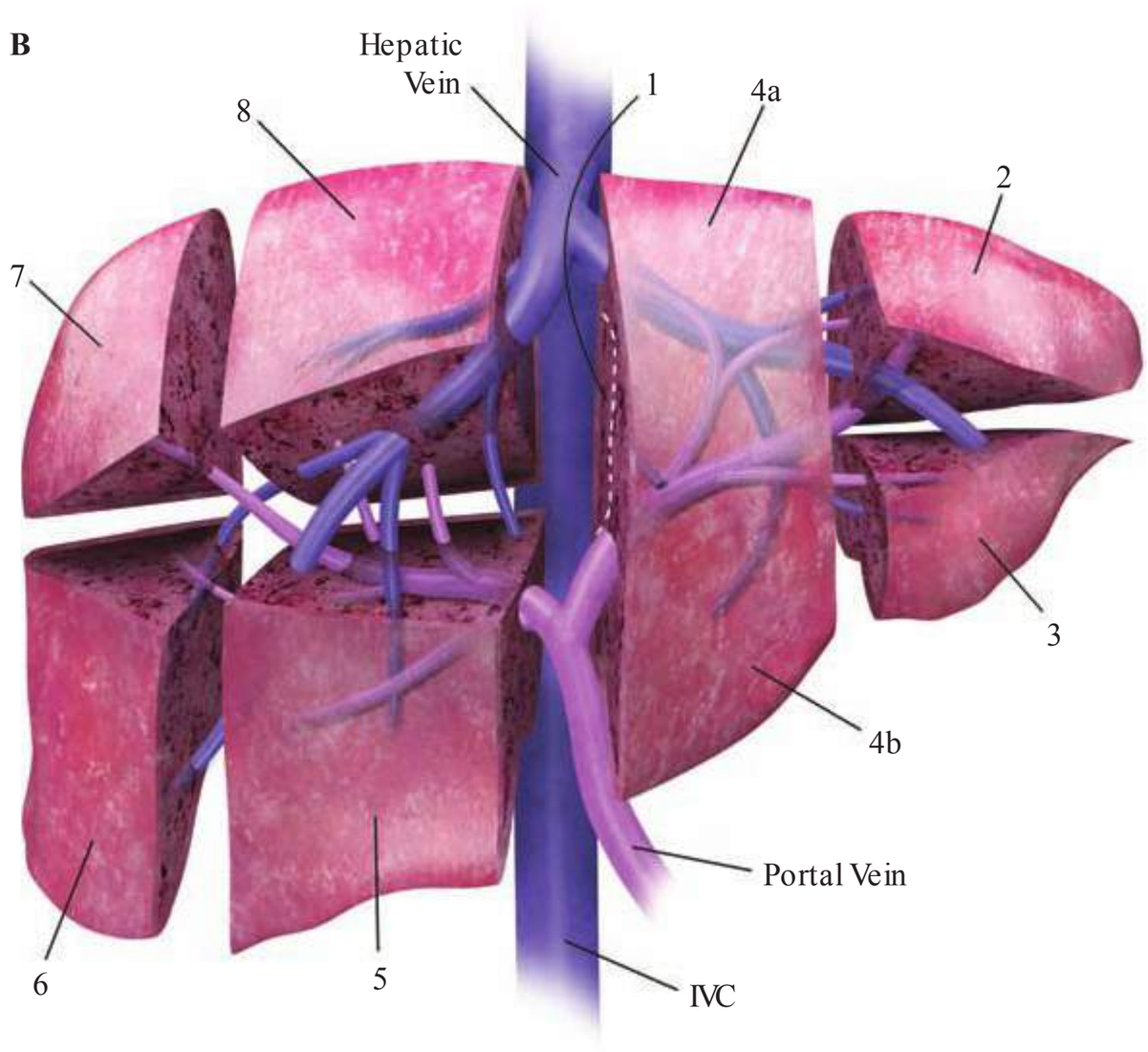


FiGUre 13-1. hepatic anatomy. (Part Breproduced, with permission, from Elsayes KM, Oldham SAA. Introduction to Diagnostic Radiology. New York, NY: McGraw-Hill Education, 2014. Figure 5.2C.)

  - Common hepatic artery divides into the hepatic artery proper and the gastroduodenal artery.
  - Hepatic artery divides into the right and left hepatic arteries.
- Venous drainage: The left, middle, and right hepatic veins drain into the inferior vena cava (IVC).
- Ligaments of the liver:
  - Falciform ligament: Connects the anterior abdominal wall to the liver; contains the ligamentum teres (obliterated umbilical vein).
  - Coronary ligament: Peritoneal reflection on the cranial aspect of the liver that attaches it to the diaphragm.
  - Triangular ligaments: The right and left lateral extensions of the coronary ligament.
  - Bare area: The posterior section of the liver against the diaphragm; has no peritoneal covering.
  - **Glisson's capsule:** The peritoneal membrane that covers the liver.
  - Cantlie's line (portal fissure): A line that passes from the left side of the gallbladder to the left side of the IVC, dividing the liver into right and left lobes. It is based on blood supply and is the basis of the four classic types of hepatic resection.
- Liver enzymes: Aspartate transaminase (AST) and alanine transaminase (ALT) are made by hepatocytes; alkaline phosphatase (alk phos) is made by ductal epithelium.

Diagnostic Imaging

Ultrasonography

- Ultrasound remains the cornerstone for diagnosing gallstones and is a great tool as it is noninvasive, fast, inexpensive, and very reliable for diagnosing gallstones.
- Its limitation is that it is user dependent.
- It should be the first test to be performed in all patients with a suspicion of gallstones.
- Can also detect masses and cysts.
- Test of choice in pregnancy.

Cholangiogram

- Provides image of the biliary tree.
- Oral and IV cholangiography—obsolete.
- T-tube cholangiogram: Performed after open exploration of the bile duct to rule out retained stones or strictures just before removal of the tube.
- Intraoperative cholangiogram: Performed on the operating table via cannulation of the cystic duct to confirm the anatomy and diagnose CBD stones.

hepatobiliary iminodiaCetiCaCid (hida) sCan

- A radionucleotide scan in which patient's own RBCs tagged with technetium-99m labeled iminodiacetic acid is injected intravenously which are taken up by the hepatocytes.
- Components in interpreting a HIDA scan
  - Normal uptake in the liver—if abnormal suggests hepatic dysfunction.
  - Uptake in the gallbladder—if absent—suggests cystic duct obstruction—cholecystitis

WARD TIP

liver blood Supply

The right and left hepatic arteries supply 50% of the liver's oxygen. The portal vein supplies the remaining 50%. However, the liver receives 75% of its blood supply from the portal vein and only 25% from the hepatic arteries. The different degrees of oxygen saturation within the arterial and venous systems account for this fact.

WARD TIP

hepatoduodenal ligament

The hepatoduodenal ligament contains the common bile duct (CBD), portal vein, and proper hepatic artery. It forms the anterior boundary of the epiploic foramen of Winslow and connects the greater and the lesser peritoneal cavities.

  - Uptake in the extrahepatic biliary tree—if absent—suggests CBD obstruction
  - Uptake in the duodenum and small intestine—if absent—suggests CBD obstruction at the level of the papilla
- A normal gallbladder would be visualized within 1 hour. Most sensitive for acute cholecystitis.
- Drawback is that it is time consuming and stones are not visualized.

Computed tomography (CT) scan

- Useful for hepatic lesions and for visualizing lymph nodes.
- Not very useful for the biliary tree.
- Expensive.
- Contraindicated in pregnancy (radiation exposure).

Endoscopic retrograde cholangiopancreatography (ERCP)

- Involves passage of a side-viewing endoscope into the duodenum, introduction of a catheter into the ampulla of Vater, and injection of contrast medium into the CBD and/or pancreatic duct; conscious sedation is necessary.
- Can be diagnostic (biopsy and show pancreatic cancer or cholangiocarcinoma) and therapeutic (remove stones, or stent an obstruction).
- One percent risk of pancreatitis from procedure.

ERCP with sphincterotomy (papillotomy)

- A cut through the sphincter of Oddi to allow the passage of stones from the CBD into the duodenum. Care to be taken to avoid injury to CBD and/or pancreatic duct.
- Often performed during ERCP but can also be performed as part of open surgery (sphincteroplasty).

Percutaneous transhepatic cholangiography (PTC)

- Involves passing a needle through the skin and subcutaneous tissues into the hepatic parenchyma and advancement into a peripheral bile duct. When bile is aspirated, a catheter is introduced through the needle and radiopaque contrast medium is injected.
- It is very useful in cases with distal obstruction where ERCP is not possible or has failed.
- It requires either USG or CT guidance.

Bile

- Constituents: Cholesterol, lecithin, bile acids, and bilirubin.
- Function: Emulsifies fats.
- Enterohepatic circulation: Bile acids are released from the **liver into the duodenum,** reabsorbed at the **terminal ileum,** and transported back to the liver via the **portal vein.**
- Cholecystokinin:
  - Released by duodenal mucosal cells.
  - Stimulates gallbladder contraction and release of bile (along with vagal stimulation).
  - Causes opening of the ampulla of Vater and slows gastric emptying.

- Is stimulated by fat, protein, amino acids, and hydrochloride (HCl).
- Trypsin and chymotrypsin inhibit its release.

Jaundice

definition

Yellowing of the skin and sclera due to an elevation in total bilirubin > 2.5. Categorized into prehepatic, hepatic, or posthepatic causes (see Table 13-1).

signs and symptoms

- Yellow skin and sclera.
- Pruritus.
- Can have hepatomegaly, tenderness of the right upper quadrant (RUQ), or signs of cirrhosis.
- Dark urine, clay-colored stools, anorexia, and nausea indicate obstructive jaundice.

diagnosis

See Table 13-1.

treatment

Treat the underlying disorder.

Liver Injury

See Table 13-2.

initial treatment

- Airway, breathing, circulation (ABCs).
- Ascertain details about mechanism of injury.

WARD TIP

Alkaline phosphatase (alk phos) is secreted by the biliary canalicular cells. In obstructive jaundice, alk phos level rises more quickly than the bilirubin level. Also, when the obstruction is relieved, the alk phos level falls more quickly than the bilirubin level.

Table 13-1. Causes of increased bilirubin

Classification	Causes	Direct Bilirubin	Indirect Bilirubin
Prehepatic	Hemolysis, Gilbert's disease, Crigler–Najjar syndrome	Normal	High
Hepatic	Alcoholic cirrhosis, acute hepatitis, primary biliary cirrhosis	High	High
Posthepatic	Gallstones, tumor	High	Normal

Table 13-2. American Association for Surgery of Trauma (AAST) Liver Injury Scale (1994 Revision)

Grade[a]	Type of Injury	Description of Injury
I	Hematoma	Subcapsular, < 10% surface area
	Laceration	Capsular tear, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, 10–50% surface area; intraparenchymal < 10 cm in diameter
	Laceration	Capsular tear, 1–3 parenchymal depth, < 10 cm in length
III	Hematoma	Subcapsular, > 50% surface area of ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma > 10 cm or expanding 3-cm parenchymal depth
	Laceration	Parenchymal disruption involving 25–75% hepatic lobe or 1–3 Couinaud's segments
IV	Laceration	Parenchymal disruption involving > 75% of hepatic lobe or > 3 Couinaud's segments within a single lobe
V	Vascular	Juxtahepatic venous injuries (i.e., retrohepatic vena cava/ central major hepatic veins)
	Vascular	Hepatic avulsion

[a]Advance one grade for bilateral injuries up to grade III

(Modified with permission, from Feliciano DV, Mattox KL, Moore EE. Trauma, 6th ed. New York: McGraw-Hill, 2008: 640. Table 32-2.)

EXAM TIP

Though the liver has been found to be protective against other organ injuries, for the same reasons (size, position), it is very vulnerable to injury itself.

- CT: Will detect blood and solid organ damage; is useful for grading injury. CT is contraindicated in the unstable or marginal patient.
- Ultrasound, if accessible, should be performed initially as focused abdominal sonography for trauma (FAST), and may then be used for serial examinations following delineation of injury on CT.

nonoperative management

EXAM TIP

Use hemodynamic stability as the deciding factor in choosing operative or nonoperative management.

- Approximately one half of patients are eligible.
- For penetrating trauma: Operative management remains standard of care.
- For blunt trauma: May attempt trial of observation if:
  - Patient is stable or stabilizes after fluid resuscitation.
  - There are no peritoneal signs.
  - The injury can be precisely delineated and graded by CT scan.
  - There are no associated injuries requiring laparotomy.
  - There is no need for excessive hepatic-related blood transfusions.
- Repeat CT scan in 2–3 days to look for expansion or resolution of injury.
- Patients may resume normal activities after 2 months.

EXAM TIP

Imaging of the liver: If contrast pool or blush is noted on CT and patient remains stable, consider an angiogram by interventional radiology for possible embolization.

operative management

- As a rule, any hemodynamically unstable patient due to a liver injury should be explored.
  - Generally needed for 20% of patients with grade III or higher injuries who present with hemodynamic instability due to hemorrhage.
- Laparotomy is undertaken through a long midline incision.

- The primary goal is the control of bleeding with direct pressure and packing.
- Patient should then be resuscitated as needed, with attention to temperature control, volume status, and acid–base balance.
- Specifics of trauma liver surgery include:
  - Pringle maneuver: Occlusion of the portal triad manually or with an atraumatic vascular clamp.
  - Finger fracture of liver to expose damaged vessels and bile ducts.
  - Debridement of nonviable tissue.
  - Placement of an omental pedicle (with its blood supply) at the site (using omentum to plug up bleeding site).
  - Closed suction drainage.
  - Major hepatic resection is indicated when the parenchyma was totally destroyed by the trauma, the extent of injury is too great for packing, the injury itself caused a near-resection, or resection is the only way to control life-threatening hemorrhage.
  - Damage control surgery: Packing the perihepatic space with a planned reoperation in 24–36 hours is indicated when the patient is severely coagulopathic, there is bilobar bleeding that cannot be controlled, there is a large expanding hematoma, other methods to control bleeding have failed, or the patient requires transfer to a level I trauma center.
- Complications:
  - Hemorrhage (5%).
  - Hemobilia (1%): Signs and symptoms—upper gastrointestinal (GI) bleed, RUQ pain, positive fecal occult blood, and jaundice.
  - Abscess.
  - Biliary fistula (7–10%): Definition—> 50 mL/d drainage for > 14 days.

OR TIP

Pringle maneuver: Occlusion of the portal triad in the hepatoduodenal ligament manually or with an atraumatic vascular clamp. Occlusion should not exceed 20 minutes if feasible.

OR TIP

If the Pringle maneuver fails to stop hemorrhage, consider an injury to the retrohepatic IVC.

Nontraumatic Liver Conditions

hepatic Abscesses

Definition

A collection of pus in the liver of bacterial, fungal, or parasitic origin that most commonly involves the right lobe. The two main subtypes are pyogenic (bacterial) and amebic.

Incidence

- Pyogenic: 8–15/100,000
- Amebic: 1.3/100,000

Risk Factors

- Pyogenic: Usually secondary to bacterial sepsis or biliary or portal vein infection; can also occur from a perforated infected gallbladder, cholangitis, diverticulitis, liver cancer, or liver metastases.
- Amebic: Patients from Central America, homosexual men, institutionalized patients, and alcoholics.

Signs and Symptoms

Fever, chills, RUQ pain, RUQ abdominal tenderness, jaundice, sepsis, and weight loss; amebic abscesses tend to have a more protracted course.

Diagnosis

- Leukocytosis.
- Elevated liver function tests (LFTs).

EXAM TIP

Watch for increasing abdominal distention with decreasing hematocrit as potential indicators of postop bleeding.

WARD TIP

In the United States, most hepatic abscesses (80%) are of bacterial origin.

WARD TIP

The most common organisms isolated from pyogenic abscesses are Escherichia coli, Klebsiella, and Proteus. Amebic abscesses are classically described as "anchovy paste" in appearance and are caused by Entamoeba histolytica, which gains access to the liver via the portal vein from intestinal amebiasis.

- Ultrasound, CT, or MRI of the liver.
- Serology for amebic abscesses.

Treatment

- Pyogenic: Ultrasound or CT-guided percutaneous drainage with IV antibiotics; operative drainage indicated if percutaneous attempts fail or cysts are multiple or loculated.
- Amebic: Operative drainage not indicated unless abscesses do not resolve with IV metronidazole or are superinfected with bacteria.

prognosis

Mortality is low for uncomplicated abscesses, but complicated abscesses carry a 40% mortality risk.

hepatiC Cysts

hydatid Cyst

Definition

A hepatic cyst caused by Echinococcus multilocularis or Echinococcus granulosus that is usually solitary and involves the right lobe of the liver.

Risk factoRs

Exposure to dogs, sheep, cattle, foxes, wolves, domestic cats, or foreign travel.

EXAM TIP

Patients with Hydatid Cysts in the liver can also have alveolar cysts in the lung (patient can have a cough, SOB, etc.); CT to confirm.

Signs anD Symptoms

Most commonly asymptomatic; can cause hepatomegaly.

Diagnosis

Often picked up incidentally on ultrasound, CT, or abdominal films, which may show calcifications outlining the cyst; eosinophilia, serology. Also will have positive Casoni skin test.

WARD TIP

Polycystic kidney disease is associated with nonparasitic cysts of the liver and berry aneurisms.

t Reatment

Never aspirate these cysts or they may spill their contents leading to anaphylactic shock. Treat with albendazole or mebendazole followed by resection.

Nonparasitic Cysts

Definition

Benign cysts within the liver parenchyma that most commonly involve the right lobe; are thought to be of congenital origin.

inciDence

Rare; 4:1 female-to-male ratio.

Signs anD Symptoms

Most cysts are small and asymptomatic; large cysts (rare) can present with increasing abdominal pain and girth and can bleed or become infected.

Diagnosis

Usually incidental; ultrasound or CT.

Treatment

Small asymptomatic cysts require no treatment; large, symptomatic cysts should be surgically treated.

Benign Liver Tumors

Cavernous Hemangioma

Definition

A benign vascular tumor resulting from abnormal differentiation of angioblastic tissue during fetal life; usually located in the right posterior segment of the liver.

Incidence

Most common tumor of the liver; occurs at all ages.

Signs and Symptoms

Usually asymptomatic; rarely presents with pain, a mass, biliary obstruction or hepatomegaly.

Diagnosis

Usually discovered incidentally; can be detected by ultrasound, CT, magnetic resonance imaging (MRI), radionuclide scan, or arteriography; **do not** biopsy, as hemorrhage can occur.

Treatment

Surgical resection if symptomatic or in danger of rupture; otherwise, observe.

WARD TIP

Kasabach–Merritt syndrome—Consumptive thrombocytopenia in patients with a hepatic hemangioma. Can lead to bleeding.

Hamartoma

Definition

- A benign focal lesion of the liver that consists of normal tissue that has differentiated in an abnormal fashion.
- Multiple subtypes, depending on the types of cells involved (e.g., bile duct hamartoma, mesenchymal hamartoma).

Incidence

Rare.

Signs and Symptoms

Typically asymptomatic; can present with RUQ pain or fullness.

Diagnosis

Usually discovered incidentally during radiologic imaging; may require histopathologic evaluation.

Treatment

Surgical excision is usually needed for diagnosis.

WARD TIP

Hepatocellular adenomas present with abdominal pain secondary to tumor rupture or bleeding in approximately one third of patients.

Hepatocellular Adenomas

Definition

A mass lesion of the liver characterized by a benign proliferation of hepatocytes.

EXAM TIP

Typical scenario: A 27-year-old female presents to her obstetrician with a history of a hepatocellular adenoma that resolved after discontinuing oral contraceptives (OCPs). She now wants to get pregnant. Does she have any specific health risks? Think: Hepatocellular adenomas treated by cessation of OCPs rather than by resection are at risk for rupture and hemorrhage during future pregnancies.

Incidence

Ninety-five percent occur in women of childbearing years.

Risk factors

OCP use, long-term anabolic steroid therapy, glycogen storage disease.

Signs and Symptoms

- Abdominal pain.
- Abdominal mass.
- Bleeding (from spontaneous rupture of large tumors).
- Can also be asymptomatic.

Diagnosis

- Ultrasound demonstrates solitary, well-demarcated, heterogenous mass.
- CT scan with IV contrast—well marginated and isoattenuating to liver. They demonstrate transient relatively homogenous enhancement returning to near isodensity on portal venous and delayed phase.

Treatment

- Cessation of OCPs
- Surgical excision is recommended as it can be a precursor for hepatocellular carcinoma.

Focal Nodular hyperplasia (FNh)

WARD TIP

Like hepatic adenomas, FNH is associated with long-term OCP use.

Definition

A benign hepatic tumor, thought to arise from hepatocytes and bile ducts, that has a characteristic "central scar" on pathologic evaluation.

Incidence

Most common in premenopausal females.

Signs and Symptoms

Usually asymptomatic; 10% of patients present with abdominal pain and/or an RUQ mass.

Diagnosis

- Usually incidental on ultrasound or CT. Classically a central scar is seen.
- Can be differentiated from hepatocellular adenoma by a Tc-99 study.

Treatment

Resect if patient is symptomatic (abdominal pain or compressive symptoms) or there is diagnostic uncertainty.

malignant liver tUmors

hepatocellular Carcinoma (hepatoma)

WARD TIP

Eighty to 90% of patients with hepatocellular carcinoma have underlying cirrhosis with alcoholic cirrhosis being the predominant type in Western countries. In the Far East posthepatitis cirrhosis is more common.

Definition

A malignant tumor derived from hepatocytes frequently found in association with chronic liver disease, particularly cirrhosis.

Incidence

- Accounts for 80% of liver cancers, but < 2% of all cancers.
- Much more common in males (3:1).
- Usually diagnosed in the fifth or sixth decade.

Risk factors

- Hepatitis B
- Hepatitis C
- Cirrhosis
- Smoking and alcohol
- Aflatoxins (found in peanuts)
- Liver flukes
- Hemochromatosis
- α_1-antitrypsin deficiency
- Anabolic steroid use
- Carbon tetrachlorides (found in cleaning agents)

Signs and Symptoms

- Weight loss
- Weakness
- Dull pain in the RUQ or epigastrium
- Nausea, vomiting
- Jaundice
- Nontender hepatomegaly
- Splenomegaly (33%)
- Ascites (50%)

Diagnosis

- Increased alk phos, AST, ALT, γ-glutamyl transferase (GGT), α-fetoprotein (AFP), and des-γ-carboxy prothrombin (DCP).
- Contrast CT and ultrasound can visualize the tumor. Enhances in the arterial phase with quick washout in the late phase and portal venous phase. Usually solitary but can be multifocal or diffuse.
- CT or ultrasound-guided needle biopsy will give the definitive diagnosis.

Treatment

- Surgical resection is the only cure, consisting of either lobectomy or segmental resection. A 1-cm margin is required.
- Transplant is also a possibility, but there often is a high recurrence rate due to the continued presence of the underlying risk factor (e.g., hepatitis B, hepatitis C, etc.). Patients are selected for liver transplantation based on the Milan criteria.
- Local chemotherapy infusion into the hepatic artery, hepatic artery embolization, and liposomal chemotherapy are the newer treatment options being explored.

Prognosis

Most patients die within the first 4 months if the tumor is not resected. After resection or transplant, the 5-year survival rate is 25–40%.

metastatic Neoplasms

Signs and Symptoms

- Patients are usually asymptomatic until the disease has become advanced and the liver begins to fail.
- Symptoms may include fatigue, weight loss, epigastric fullness, dull RUQ pain, ascites, jaundice, or fever.

Diagnosis

- Increased alk phos, GGT, lactic dehydrogenase (LDH), AST, and ALT (nonspecific).
- Metastases will enhance on contrast CT (see Figure 13-2).
- Intraoperative ultrasound with liver palpation is the most sensitive diagnostic tool.

WARD TIP

A bruit can commonly be heard over a hepatocellular carcinoma due to its abundant vascularity.

WARD TIP

milan criteria:

- Single tumor with diameter ≤ 5 cm, or up to 3 tumors each with diameter ≤ 3 cm.

WARD TIP

Why do ascites and anasarca occur in liver disease? Because the liver makes albumin, which is necessary to generate oncotic pressure and maintain fluids in the vasculature.

WARD TIP

The most common hepatic malignancy is metastases. The primary is usually from colon, breast, stomach, or lung, with bronchogenic carcinoma being the most common primary cancer.

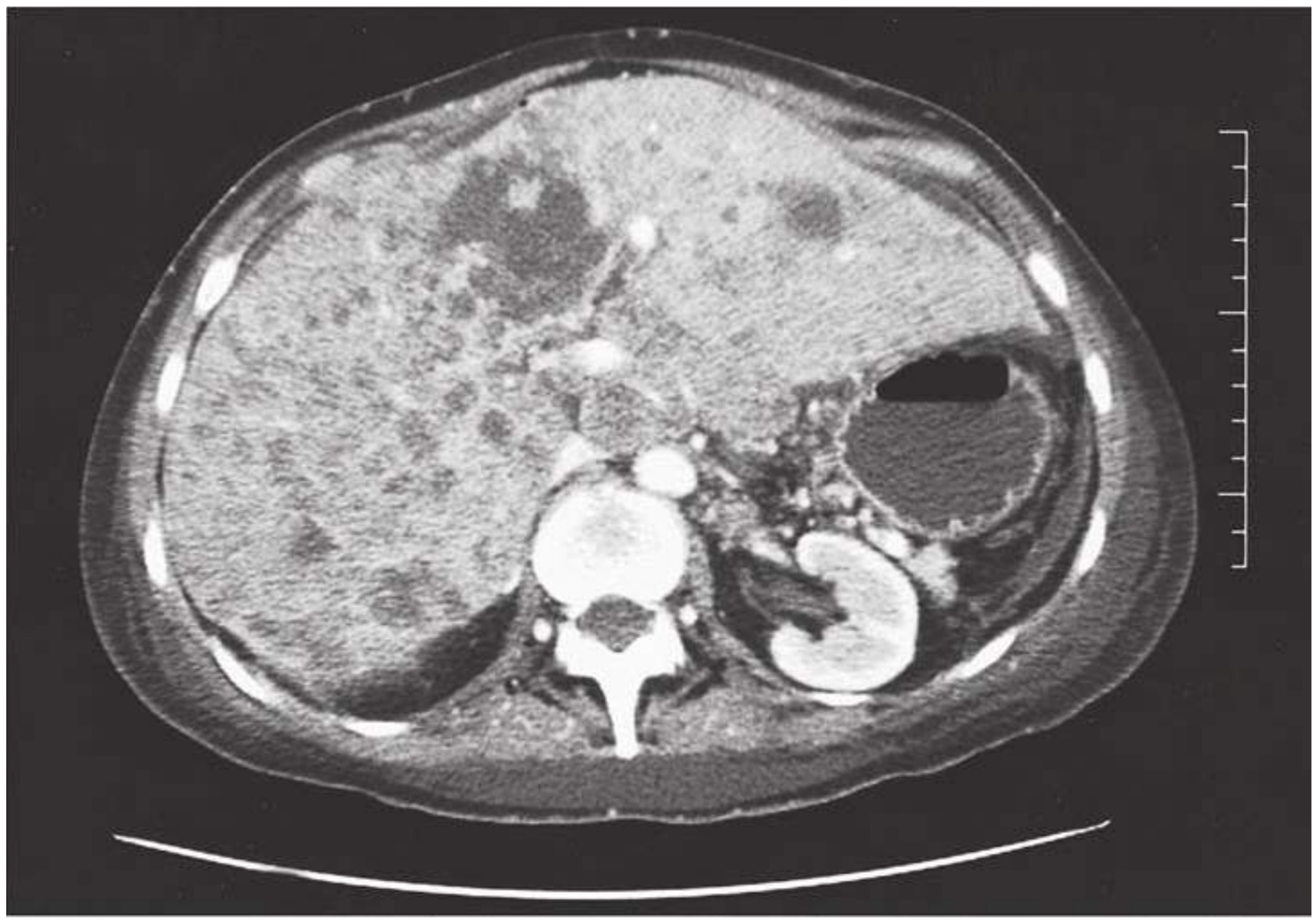


FIGURE 13-2. Abdominal CT in hepatic metastasis. Note slitlike appearance of intrahepatic IVC secondary to compression by metastasis. Also note presence of ascites and bilateral pleural effusions, left greater than right.

Treatment

- Resection, if possible, is the treatment of choice.
- Radiofrequency ablation (RFA) is now extensively done even for multiple lesions, and the short-term results are promising.

Liver Failure

Child–Pugh Score

Child's classification estimates hepatic reserve in patients with liver failure (see Table 13-3).

TABLE 13-3. Child–Pugh Score for Liver Failure

	NUMBER OF POINTS ACCORDED		
VARIABLE	**1**	**2**	**3**
Bilirubin (mg/dL)	< 2.0	2.0–3.0	> 3.0
Albumin (mg/dL)	> 3.5	2.8–3.5	< 2.8
Ascites (clinical evaluation)	None	Easily controlled	Poorly
Neurologic disorder	None	Minimal	Advanced
Prothrombin time (sec < control)	< 4.0	4.0–6.0	> 6.0

Correlation with Child's class: 5–6 points, Class A; 7–9 points, Class B; 10–15 points, Class C.

The Model for End-Stage Liver Disease (MELD) score is used for liver transplant allocation in the United States. The MELD formula is dependent on INR, SCr (serum creatinine level in milligrams per deciliter), and Tbil (total serum bilirubin level in milligrams per deciliter).

MELD Score = 9.57 Ln(SCr) + 3.78 Ln(Tbil) + 11.2 Ln(INR) + 6.43

where Ln represents natural logarithm.

Portal Hypertension

definition

Portal pressure > 10 mmHg (measure with indirect hepatic vein wedge pressure).

Causes

- Prehepatic: Congenital atresia, cyanosis, or portal vein thrombosis.
- Intrahepatic: Cirrhosis, hepatic fibrosis from hemochromatosis, Wilson's disease, or congenital fibrosis.
- Posthepatic: Budd–Chiari syndrome (thrombosis of the hepatic veins), hypercoagulable state, lymphoreticular malignancy.

signs and symptoms

- Jaundice
- Splenomegaly
- Palmar erythema
- Spider angiomata
- Ascites
- Truncal obesity with wasting of the extremities
- Asterixis (a flapping hand tremor)
- Hepatic encephalopathy

portosystemic Collaterals and their Clinical manifestations

- Left gastric vein to the esophageal veins—esophageal varices.
- Umbilical vein (via the falciform ligament) to the epigastric veins—caput medusa.
- Superior hemorrhoidal vein to the middle and inferior hemorrhoidal veins—hemorrhoids.
- Veins of Retzius (posterior abdominal wall veins) to the retroperitoneal lumbar veins—retroperitoneal varices.

Diagnosis

- Suggestive history and physical examination.
- Duplex Doppler ultrasound: Initial procedure of choice.
  - CT scan of the abdomen—Cirrhosis of the liver, splenomegaly, engorged and tortuous collaterals and varices, prominent abdominal wall and retroperitoneal veins.
- Venous phase of visceral angiography: Defines portal anatomy more precisely.

Treatment

- Aimed at reducing portal pressure.

WARD TIP

The most common cause of portal hypertension in the United States is cirrhosis from alcoholism. The most common cause outside North America is schistosomiasis.

WARD TIP

Rule of two thirds for portal hypertension: Two thirds of patients with cirrhosis develop portal hypertension. Two thirds of patients with portal hypertension develop esophageal varices. Two thirds of patients with esophageal varices will bleed from them. Note: Only 10–15% of alcoholics develop cirrhosis.

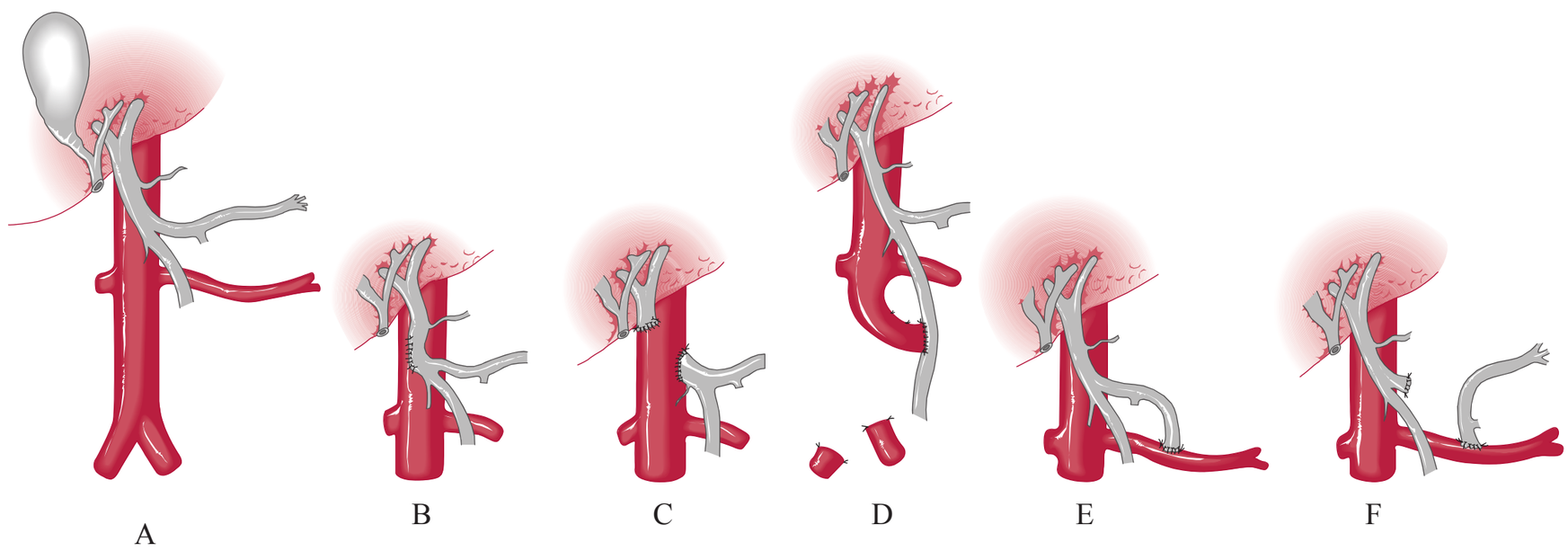


FIGURE 13-3. Types of portacaval anastomoses: a: Normal. b: Side-to-side. C: end-to-side. D: mesocaval. e: Central splenorenal. F: Distal splenorenal (Warren). The H-mesocaval shunt is not illustrated. (Reproduced, with permission, from Doherty GM. Current Diagnosis & Treatment: Surgery. 14th ed. New York, NY: McGraw-Hill Education; 2015. Figure 24-8.)

- Shunts (see Figure 13-3):
  - **Splenorenal (Warren shunt):** Connects the splenic vein to the left renal vein. Used for patients with esophageal varices and a history of bleeding.
  - **End to side:** Connects the end of the portal vein to the side of the IVC. This is considered a total shunt.
  - **Side to side:** Connects the side of the portal vein to the side of the IVC. This is considered a partial shunt.
  - **Portacaval H graft:** A synthetic graft is attached from the portal vein to the IVC. This is considered a partial graft.
  - **Mesocaval H graft:** A synthetic graft is attached from the superior mesenteric vein (SMV) to the IVC.
- **Complications:** Increased incidence of hepatic encephalopathy because more toxins are diverted to the systemic circulation (except for the Warren shunt), and death from hepatic failure due to decreased blood flow to the liver.
- **Liver transplant:** Ideal candidate is a young patient with cirrhosis and an episode of bleeding from esophageal varices.
- Medical treatment: Beta blockers to reduce portal pressure.

WARD TIP

Splenomegaly is the most common clinical finding in portal hypertension. Removal is almost never warranted.

Morbidities Resulting from Liver Failure

hepatiCenCephalopathy

Definition

- Altered mental status due to hepatic insuffciency.
- Toxins that are normally cleared by the liver are retained in the circulation.
- The exact toxin that causes the central nervous system (CNS) changes is unknown but has been theorized to be ammonia, γ-aminobutyric acid (GABA), mercaptans, or short-chain fatty acids.

epidemiology

- Occurs in all cases of fulminant hepatic failure.
- Occurs in one half of patients with end-stage liver disease requiring transplantation.
- Occurs in one third of patients with cirrhosis.

Cause

Precipitating factors include:

- Infection (watch for spontaneous bacterial peritonitis [SBP]).
- Potassium, magnesium, or other electrolyte depletion.
- Use of opiates, sedatives, or other hepatically cleared drugs.
- GI bleed.
- Excess dietary protein.

Signs and Symptoms

- Change in level of consciousness, mood disturbance, decreased attention span.
- Lethargy, coma.
- Normal electroencephalogram (EEG).
- Asterixis flapping tremor of hand.

Diagnosis

An elevated serum ammonia level along with signs of altered mental status and altered LFT is diagnostic.

Treatment

Lactulose and neomycin (PO or PR) to reduce intestinal formation and absorption of ammonia.

WARD TIP

In hepatic encephalopathy, the serum ammonia level **does not** correlate with the degree of encephalopathy.

ascites

Definition

- Excess fluid in the peritoneal cavity.
- Sodium and fluid retention by the kidney, low plasma oncotic pressure due to low albumin production by the failing liver, and elevated hydrostatic pressure in the hepatic sinusoids or portal veins cause fluid to be lost into the peritoneal cavity.
- Ascitic fluid is frequently the site of SBP in patients with liver disease.

Signs and Symptoms

Distended abdomen, fluid wave, shifting dullness.

Treatment

- Reduce sodium intake.
- Potassium-sparing diuretic (e.g., spironolactone).
- Abdominal paracentesis.
- Removing too much ascitic fluid or removing the fluid too quickly will cause intravascular fluid to be drawn into the peritoneal cavity. This leads to a loss of intravascular volume and can cause hypovolemic shock.

EXAM TIP

A **Leveen shunt** is a peritoneal-jugular shunt used to decrease ascites. One drawback is that it may increase hepatic encephalopathy.

esophageal varices

Definition

Engorged esophageal or gastric veins, usually resulting from increased portal pressure from liver disease.

Signs and Symptoms

- Asymptomatic unless rupture occurs.
- With rupture: Upper GI bleeding with hematemesis, melena, and/or hematochezia.

Diagnosis

Esophagogastroduodenostomy (EGD).

Treatment of r uptured Varices

Options include:

- Endoscopic sclerotherapy.
- Vasopressin or somatostatin injection.
- Balloon tamponade (Sengstaken–Blakemore tube).
- Transjugular intrahepatic portacaval shunt (TIPS): This is used more for long-term management, rather than during an acute bleed; it shunts pressure away from the esophageal vessels. Also useful in patients awaiting a liver transplant with advanced cirrhosis and portal hypertension.
- Intraoperative placement of a portacaval shunt.
- Liver transplant.
- β-blockers can be used to decrease portal pressure and reduce the incidence of rupture.

prognosis

Poor, even with treatment. Ruptured esophageal varices have a 50% death rate.

OR TIP

Calot's triangle: Inferior border of the liver, common hepatic duct, and cystic duct. The cystic artery runs through it, and the associated lymph node is called Calot's node. The right hepatic artery is adjacent to the cystic duct in Calot's triangle and, as such, is susceptible to injury during cholecystectomy.

CapUt medUsa

Def nition

Engorged abdominal wall veins—a sign of increased portal pressure.

Signs and Symptoms

Mass of veins extending from around the umbilicus, periumbilical bruit (Cruveilhier–Baumgarten bruit).

The Biliary System

anat omy of t he bil iar y t r ee

See Figure 13-4.

- Intrahepatic ducts converge to become the right and left hepatic ducts.
- Right and left hepatic ducts converge, forming the common hepatic duct.
- Cystic duct comes off the gallbladder and joins the common hepatic duct to become the CBD.
- CBD empties into the duodenum via the ampulla of Vater.

anat omy of t he gall bl adder

WARD TIP

The infundibulum of the gallbladder is also called **h artman's pouch.**

- The proximal end of the gallbladder near the cystic duct is called the infundibulum, and the larger distal end of the gallbladder is called the fundus.
- The valves within the cystic duct are called the spiral valves of Heister.
- The gallbladder collects bile directly from the liver via small bile ducts called ducts of Luschka.

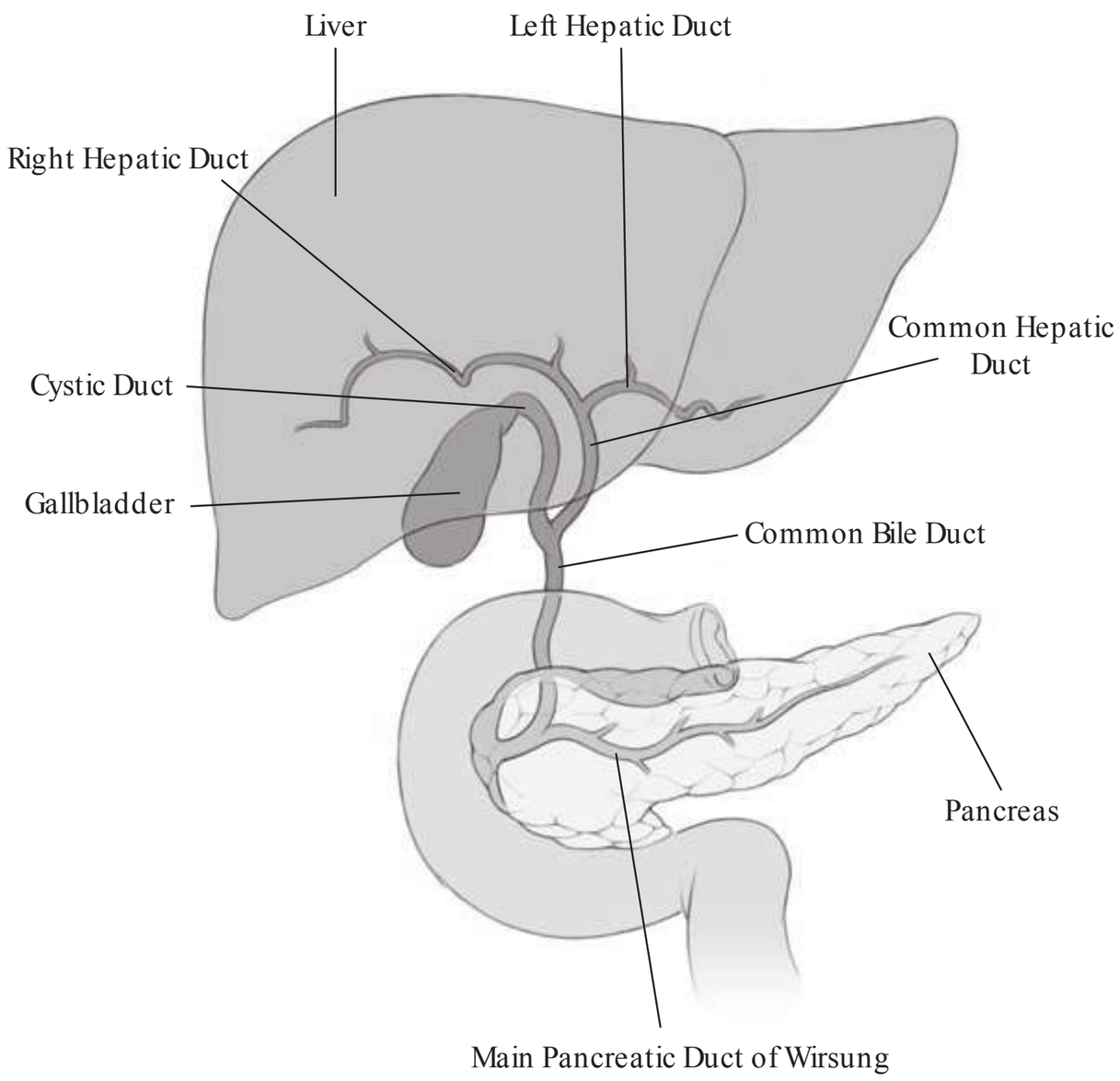


FiGUre 13-4. anatomy of the biliary tree.

Conditions of the Gallbladder and the Biliary Tree

Cholelithiasis

Definition

Stones in the gallbladder formed from an imbalance between the contents of bile—cholesterol, bile salts, and bile pigments. Eighty-five percent of stones are composed primarily of cholesterol, while the remaining 15% are pigmented.

incidence

Approximately 10% of the U.S. population has gallstones.

risk Factors

- The typical ones are: **F**emale, **f**at, **f**ertile, and **f**orty.
- Other risk factors include Native American race, pregnancy, OCPs, Western diet, inflammatory bowel disease (IBD), hyperlipidemia, ileal resection (due to loss of enterohepatic circulation), and total parenteral nutrition (due to prolonged cholestasis).

Signs and Symptoms

- Most patients are asymptomatic.
- Symptomatic patients classically complain of severe RUQ pain that radiates to the back, epigastrium, or left upper quadrant (LUQ) that tends to be worse after eating (especially after fatty foods) and may be associated with nausea and vomiting.
- The symptom complex is called biliary colic and typically resolves over a few hours.

WARD TIP

Only 15% of gallstones have enough calcium to be radiopaque. The majority of kidney stones, however, have sufficient calcium to be radiopaque on plain films.

WARD TIP

Risk factors for cholelithiasis:

- **F**emale
- **F**at
- **F**ertile
- **F**orty
- **F**latulent
- **F**amilial
- **F**ibrosis, cystic
- **F**-Hgb (sickle cell disease)



FiGUre 13-5. Sonogram demonstrating classic "headlight" appearance of cholelithiasis.

EXAM TIP

mirizzi's syndrome is external compression of the common hepatic duct by a gallstone impacted in the cystic duct.

WARD TIP

hydrops of the gallbladder: Complete obstruction of the cystic duct by a gallstone, causing the gallbladder to fill with fluid from the gallbladder mucosa. The fluid is often milky white as the bile pigments are absorbed due to chronic obstruction.

Diagnosis

- Often incidental, as most patients are asymptomatic.
- Abdominal plain films pick up 15% of gallstones.
- Ultrasound: Procedure of choice; classic findings include an acoustic shadow ("headlight") and gravity-dependent movement of gallstones with patient repositioning (see Figure 13-5).

Treatment

Asymptomatic cholelithiasis does not require cholecystectomy unless the patient:

- Has a porcelain gallbladder (which has an increased incidence of carcinoma).
- Has sickle cell anemia.
- Has a stone > 2–3 cm.
- Is a pediatric patient.
- Is immunocompromised.

Symptomatic cholelithiasis requires cholecystectomy. A laparoscopic cholecystectomy can be performed on 95% of patients. Medical treatment of cholelithiasis involves chenodeoxycholic acid or ursodeoxycholic acid drugs.

Cholecystitis

acute Cholecystitis

Definition

Inflammation of the gallbladder wall, usually due to obstruction of the cystic duct by gallstones.

Signs and Symptoms

- RUQ tenderness and guarding are present, usually for > 3 hours.
- Fever, nausea, vomiting, and anorexia are nonspecific and variable.
- Murphy's sign: Pain on deep inspiration resulting in inspiratory arrest (positive in about one third of patients) upon palpation of RUQ.

WARD TIP

In acute cholecystitis, the pain is similar in character to cholelithiasis but typically lasts longer (> 3 hours).

- Sonographic Murphy's: Pain over RUQ when palpated with ultrasound probe (87% sensitivity).
- One third of patients develop exquisitely tender RUQ mass late in course.

Diagnosis

- Labs: Leukocytosis with or without increased alk phos LFTs, amylase, and total bilirubin.
- Ultrasound: Reveals inflammation of the gallbladder wall (> 4 mm), pericholecystic fluid and stones in the gallbladder. Positive predictive value of all three is 90%. Will also see dilation of the CBD if a stone is lodged in CBD.
- HIDA scan (most sensitive): Non-filling of the gallbladder even when the small bowel is visualized is characteristic of acute cholecystitis (see Figures 13-6 and 13-7).

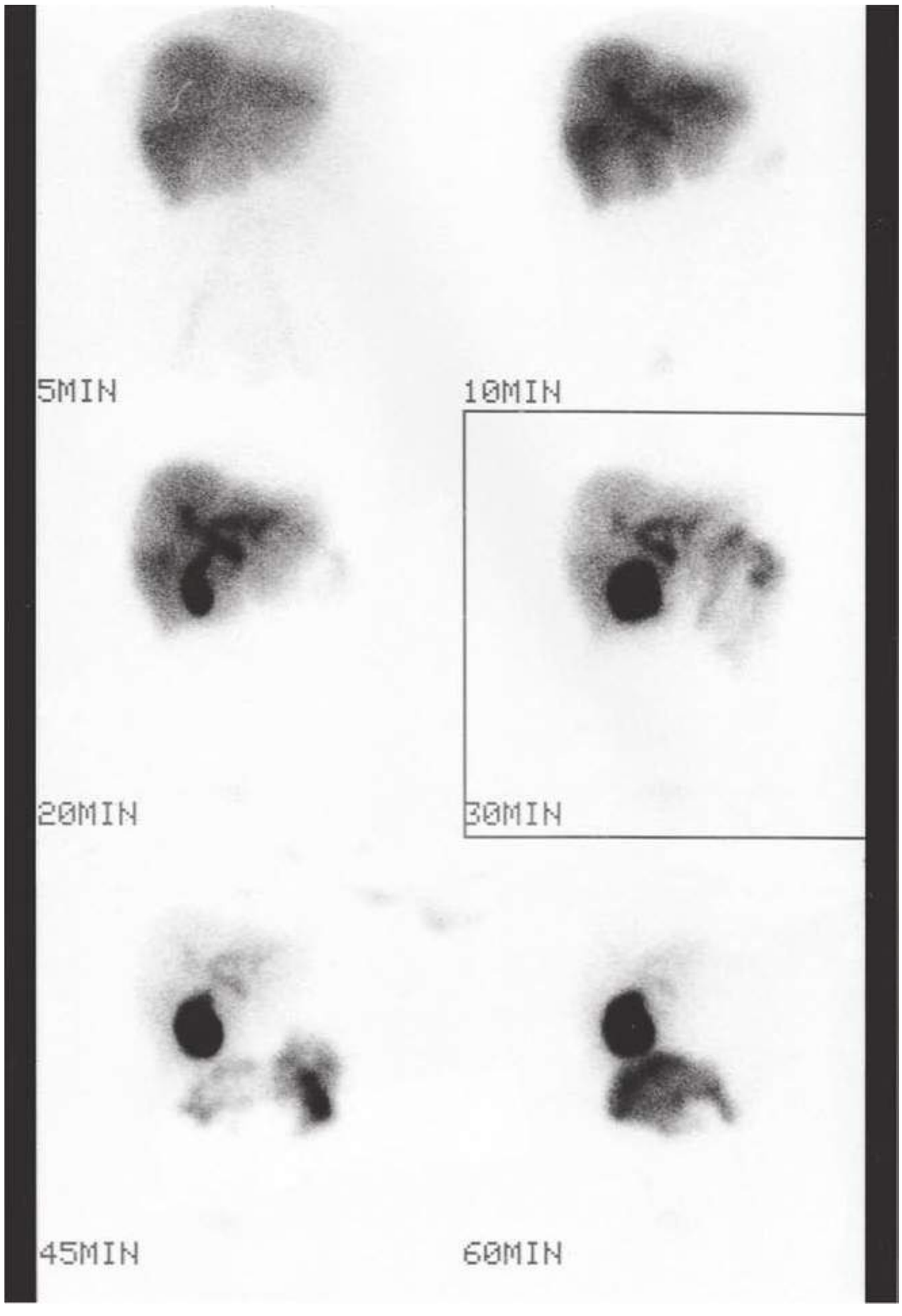


FIGURE 13-6. Normal hiDa scan, demonstrating uptake of contrast in intrahepatic bile ducts and gallbladder at 20 minutes and excretion into small bowel at 30 minutes.

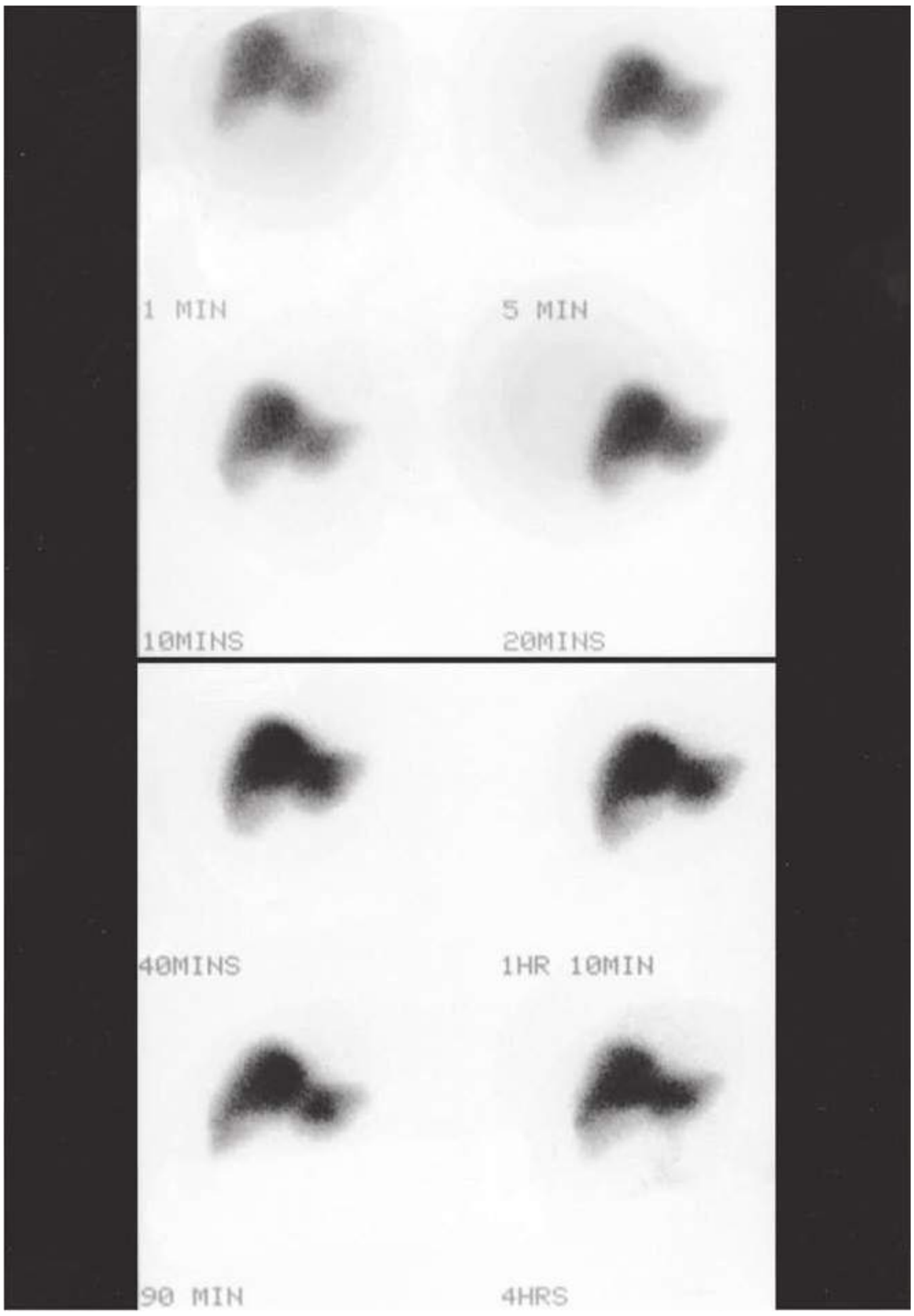


FIGURE 13-7. HIDA scan, consistent with acute cholecystitis. The gallbladder is not visualized even at 4 hours, even though the small bowel is.

Treatment

- NPO.
- IV fluids.
- IV antibiotic (broad spectrum).
- IV analgesia.
- Cholecystectomy within 24–48 hours.
- Often done laparoscopically; if inflammation prevents adequate visualization of important structures, convert to open cholecystectomy.

Emphysematous Cholecystitis

See Figures 13-8 and 13-9.

- Severe variant of cholecystitis caused by gas-forming bacteria.
- Relatively rare.

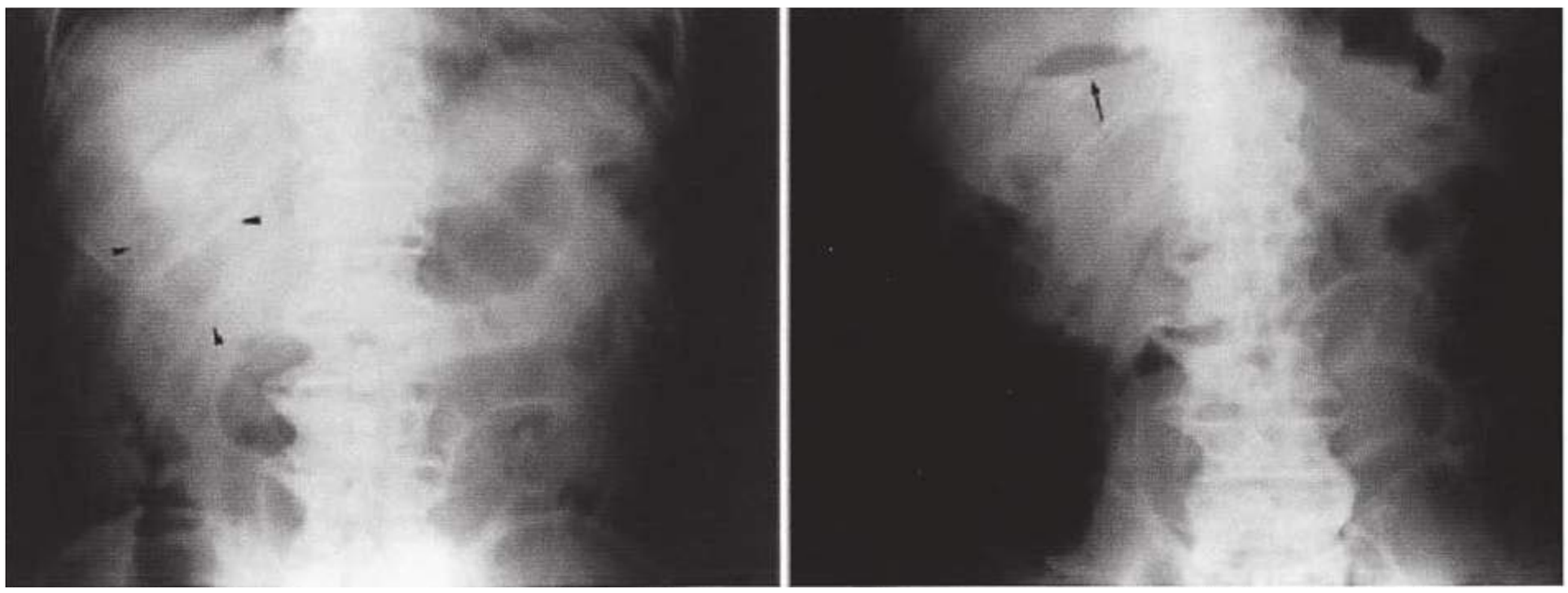


FIGURE 13-8. aXr demonstrating rimlike gas within gallbladder. (Reproduced, with permission, from Ise N, et al. Acute emphysematous cholecystitis preceded by symptoms of ileus: report of a case. Surg Today 32:183–185, 2002.)

- Often results in perforation of the gallbladder, high mortality and morbidity.
- Typically affects elderly diabetic men.

acalculous Cholecystitis

Definition

Acute cholecystitis without evidence of gallstones; thought to be due to biliary stasis and ischemia (low perfusion states).

inciDence

Ten percent of cases of acute cholecystitis.

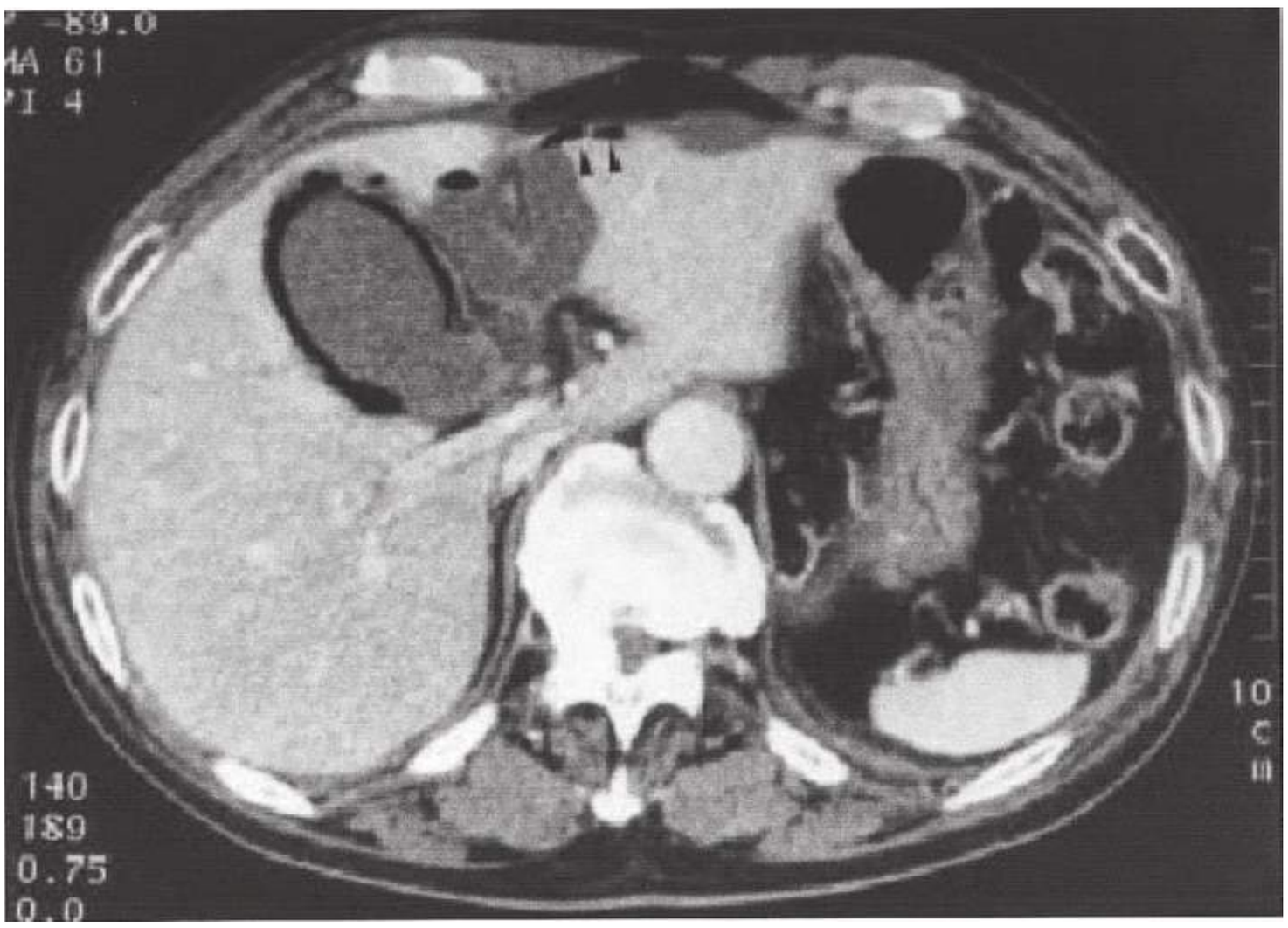


FIGURE 13-9. abdominal CT shows air in the lumen and within the wall of the gallbladder, surrounding fatty infiltration and intraperitoneal gas. (Reproduced, with permission, from Ise N, et al. Acute emphysematous cholecystitis preceded by symptoms of ileus: report of a case. Surg Today 32:183–185, 2002.)

Risk factors

Most often seen in intensive care unit (ICU) patients with multiorgan system failure, trauma (especially after major surgery), burns, sepsis, and TPN.

Diagnosis

- Labs: Leukocytosis, with or without increased alk phos LFTs, amylase, and total bilirubin.
- Ultrasound: Biliary sludge and inflammation; can also be used to detect complications (e.g., gangrene, empyema, or perforation of the gallbladder).
- HIDA scan: To confirm diagnosis.

Treatment

- Urgent cholecystectomy; percutaneous cholecystostomy is an option in patients with high surgical risk.
- Cholecystectomy:
  - Surgical removal of the gallbladder.
  - May be done open or via laparoscopy.
  - Open is done by Kocher's incision (right subcostal).
  - Laparoscopic is by 4-port technique (umbilical 10-mm, subxiphoid 10-mm and two 5-mm ports).
  - Safe dissection of the Calot's triangle is key to demonstrate critical view of safety.
  - If the whole GB cannot be safely removed partial cholecystectomy should be done.

WARD TIP

Ten percent of cases of acute cholecystitis are not associated with gallstones. This is referred to as **acalculous cholecystitis.**

Choledocholithiasis

Definition

Stones in the CBD. This can, but does not necessarily, cause cholecystitis.

Incidence

Found in 6–15% of acute calculous cholecystitis and 1–2% of acalculous cholecystitis at surgery.

Signs and Symptoms

Epigastric or RUQ pain and tenderness, jaundice, cholangitis, or recurrent attacks of acute pancreatitis without other known risk factors.

Diagnosis

- Labs: Increased alk phos LFTs, and total and direct bilirubin.
- ERCP: Gold standard for diagnosis of CBD stones; also provides a therapeutic option (see below).
- Endoscopic ultrasound: Less sensitive than ERCP but also less invasive; more sensitive than transabdominal ultrasound.
- Transabdominal ultrasound: Highly specific but not very sensitive for CBD stones.
- CT scan or MRI/MRCP is also very useful for evaluating CBD obstruction and biliary anatomy.

Treatment

- ERCP: Involves endoscopic sphincterotomy with retrieval of the CBD stone(s) with a basket (85–90% successful).

WARD TIP

Ascending cholangitis is a life-threatening emergency. It can be precipitated by any irritation or obstruction of the biliary tree.

EXAM TIP

Charcot's triad:

- RUQ pain
- Fever
- Jaundice

- If ERCP fails, the CBD can be opened surgically and the stones removed. Intraoperative cholangiogram has to be done to evaluate for underlying strictures. A T-tube is placed so bile can drain externally. It is removed 2–3 weeks later on an outpatient basis.

Cholangitis

Acute (Ascending) Cholangitis

Definition

Bacterial infection of the bile ducts usually associated with obstruction of the CBD—gall stones (most common), strictures, or mass.

Signs and Symptoms

- Fever, chills.
- Nausea, vomiting.
- Abdominal pain with or without altered mental status and septic shock.

Diagnosis

- Labs: Leukocytosis with increased bilirubin, alk phos and LFTs.
- Ultrasound: Should be the initial study; dilation of common and intrahepatic bile ducts along with gallstones, and a thickened, edematous gallbladder wall.
- CT scan with contrast or MRI/MRCP can be done—shows hyperemic wall of the CBD with ductal dilatation and cut-off point suggesting site of obstruction.
- ERCP/PTCA: Provides a definitive diagnosis; can also be therapeutic.
- Bile cultures: Obtain to facilitate proper antibiotic treatment; offending organisms are usually enteric gram negatives and enterococci.

Treatment

- NPO, IV fluids, and broad spectrum IV antibiotics. Close monitoring in ICU.
- If patient is in shock, decompress bile duct and remove obstruction immediately by ERCP/PTC. If unsuccessful, intraoperative decompression with T-tube placement is indicated.
- If the patient is stable, continue conservative management with definitive treatment later.

Sclerosing Cholangitis

Definition

A chronic, progressive inflammatory process of the biliary tree of unknown etiology that results in strictures and, in most cases, leads to cirrhosis and liver failure. Associated with autoimmune phenomena, particularly ulcerative colitis.

Incidence

Two-to-one male predominance with median age of onset at 40 years.

Risk Factors

IBD (ulcerative colitis), pancreatitis, diabetes, trauma to the common hepatic duct.

EXAM TIP

Reynolds' pentad:
- Charcot's triad plus
- Central nervous system (CNS) symptoms
- Septic shock

EXAM TIP

Common causes of CBD obstruction:
SiNGe

Stricture
iatrogenic causes (ERCP/PTC or biliary stent placement)
Neoplasm
Gallstones
extrinsic compression (e.g., pancreatic pseudocyst/pancreatitis)

WARD TIP

Seventy percent of patients with sclerosing cholangitis have IBD, whereas 3–7.5% of patients with IBD have sclerosing cholangitis.

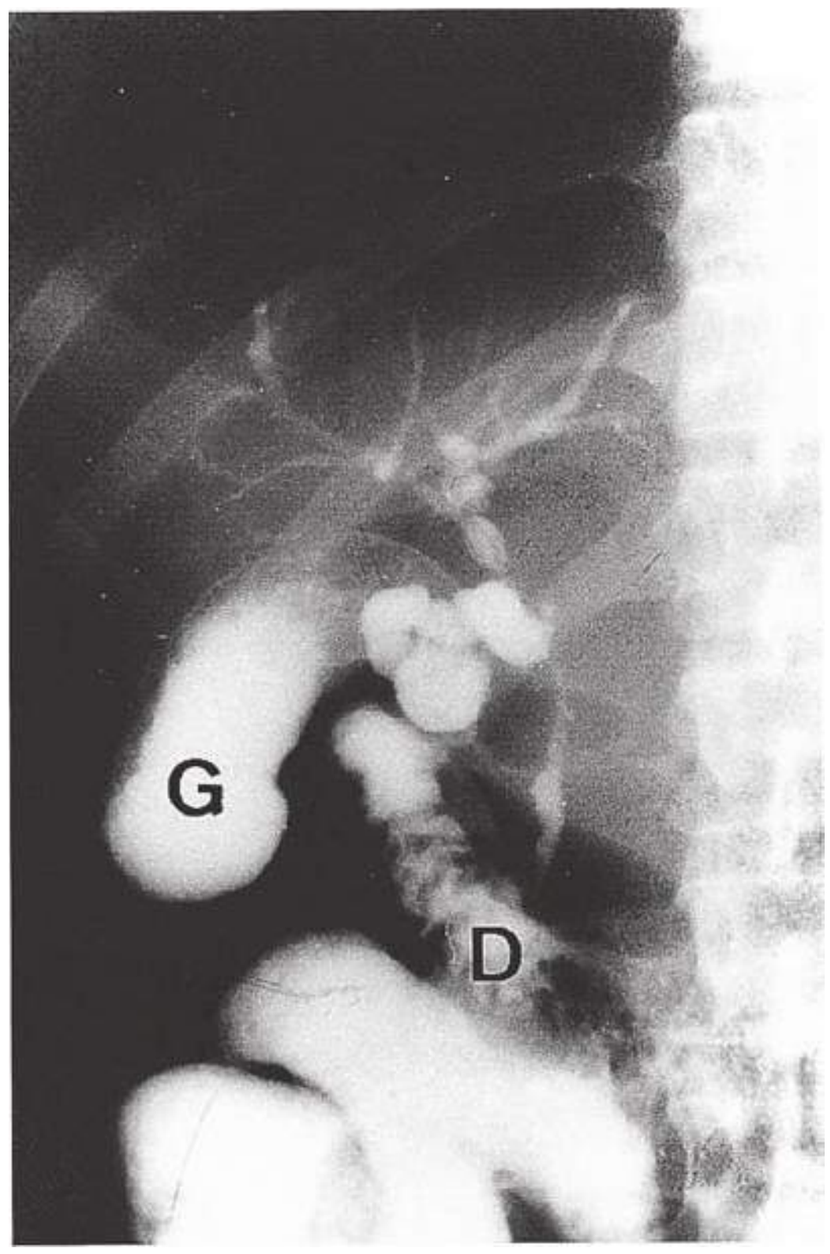


FIGURE 13-10. ERCP demonstrating "beads on a string" appearance of bile ducts in sclerosing cholangitis. G, gallbladder; D, duodenum. (Reproduced, with permission, from Nakanuma Y, et al. Definition and pathology of primary sclerosing cholangitis. J Hepatobiliary Pancreat Surg 6:333–342, 1999.)

WARD TIP

Complications of sclerosing cholangitis:
- Cirrhosis
- Cholangitis
- Obstructive jaundice
- Cholangiocarcinoma

EXAM TIP

Typical scenario: A 75-year-old female with a past history of cholelithiasis presents complaining of RUQ pain that radiates to her back, with nausea, vomiting, and abdominal distention. Abdominal plain films show air in the biliary tree and a "stepladder" appearance of the small bowel. Think: The history is consistent with both cholelithiasis and small bowel obstruction, and findings on abdominal radiograph are suggestive of gallstone ileus.

Signs and Symptoms

Many patients are asymptomatic at the time of diagnosis, but symptoms can include fever, weight loss, fatigue, pruritus, jaundice, hepatomegaly, splenomegaly, and hyperpigmentation.

Diagnosis

ERCP/PTC reveal a "beads on a string" appearance of the bile ducts (see Figure 13-10). alk phos is almost always elevated.

Treatment

- Balloon dilation with stent placement can be performed for palliative purposes, but definitive treatment varies depending on the location of the strictures.
- Extrahepatic strictures: Hepatoenteric anastomosis with removal of the extrahepatic ducts and T-tube placement for external drainage of bile.
- Intrahepatic strictures: Liver transplant.

Prognosis

- Ten percent of patients develop cholangiocarcinoma.
- Ten-year survival is 75%.

GALLSTONE ILEUS

Definition

- Small bowel obstruction caused by a gallstone; the ileocecal valve is the most common site of obstruction.

- Most often, a large stone has eroded a hole through the gallbladder wall to the duodenum, causing a cholecystoenteric fistula. A gallstone escapes through this hole into the GI tract and eventually gets stuck in the ileum, causing small bowel obstruction.

Incidence

Most common in women over 70.

Signs and Symptoms

Symptoms of acute cholecystitis followed by signs of small bowel obstruction (nausea, vomiting, abdominal distention, RUQ pain).

Diagnosis

- Abdominal plain films: May show the pathognomonic features of pneumobilia, dilated small bowel, and a large gallstone in the right lower quadrant (RLQ).
- Ultrasound: Useful to confirm cholelithiasis; may also identify the fistula.
- Upper and lower GI series: Other diagnostic options that are usually unnecessary.
- CT scan of the abdomen: Shows proximally dilated small bowel with a transition point (stone in the lumen) and collapsed distal loops. The cholecystoenteric fistula can also be visualized.

Treatment

Exploratory laparotomy, removal of the gallstone (via proximal longitudinal enterotomy and milking the stone out, with repairing it transversely), and possible small bowel resection. Cholecystectomy and fistula repair can be undertaken if the patient is hemodynamically stable or can be performed in another setting.

OR TIP

The whole small bowel must be palpated in Gallstone ileus to detect other gallstones in the GI tract.

Carcinoma of the gallbladder

Definition

Malignant neoplasm of the gallbladder, the majority of which are adenocarcinomas.

Incidence

Extremely rare (< 1% of patients with cholelithiasis); incidence increases with age with a peak at 75 years; female-to-male ratio 3:1.

Risk Factors

Include porcelain gallbladder, gallstones, choledochal cysts, gallbladder polyps, and typhoid carriers with chronic inflammation.

Signs and Symptoms

Most patients are asymptomatic until late in the course when findings may include abdominal pain, nausea, vomiting, weight loss, RUQ mass, hepatomegaly, or jaundice. Gallbladder carcinoma tends to metastasize earlier in the course.

Diagnosis

Ultrasound, CT, MRI, or ERCP/PTC.

WARD TIP

Courvoisier's sign: A palpable, nontender gallbladder often associated with cancer in the head of the pancreas or the gallbladder.

Treatment

- Varies depending on the extent of tumor involvement.
- Tumor confined to gallbladder mucosa: Cholecystectomy.
- Tumor involving muscularis or serosa: Radical cholecystectomy, wedge resection of overlying liver (segment IVb and V), and portal lymph node dissection.
- Tumor involving liver: Consider palliative measures such as decompression of the proximal biliary tree or a bypass procedure of the obstructed duodenum.

Benign Tumors of the Bile Ducts

Definition

Tumors that arise from ductal glandular epithelium most commonly found at the ampulla of Vater; most are adenomas, of polypoid morphology, and < 2 cm in size.

Signs and Symptoms

Intermittent jaundice and RUQ pain.

Diagnosis

Intraoperative cholangiogram, ultrasound, ERCP/PTC.

Treatment

Resection of the tumor with a margin of duct wall either intraoperatively or endoscopically.

Choledochal Cysts

Abnormal cystic dilatations of intrahepatic and/or extrahepatic bile ducts. Can be congenital, acquired, or familial.

- Incidence: In Western population—1:100,000 to 1:150,000.
- Pathogenesis: Exact etiology is unknown, but an abnormal pancreatobiliary junction appears to play an important role.

Presentation

- Can be asymptomatic and be incidentally diagnosed on cross-sectional abdominal imaging.
- Sometimes can be symptomatic—abdominal pain, cholangitis, jaundice. Classic presentation—abdominal pain, jaundice, and a palpable abdominal mass.

Classification

- Todani classification of biliary cysts (Figure 13-11)
- Type I—Fusiform dilatation of the extrahepatic bile duct.
- Type II—Saccular outpouching from the extrahepatic bile duct—bile duct diverticulum.
- Type III—Choledochocele—cystic dilatation of the intraduodenal portion of the bile duct.
- Type IVa—Fusiform dilatation of both extrahepatic and intrahepatic bile ducts.
- Type IVb—Fusiform dilatation of only the extrahepatic bile ducts.
- Type V—Cystic dilatations of intrahepatic ducts only—Caroli's disease.

Diagnosis

USG, CT, MRI, ERCP.

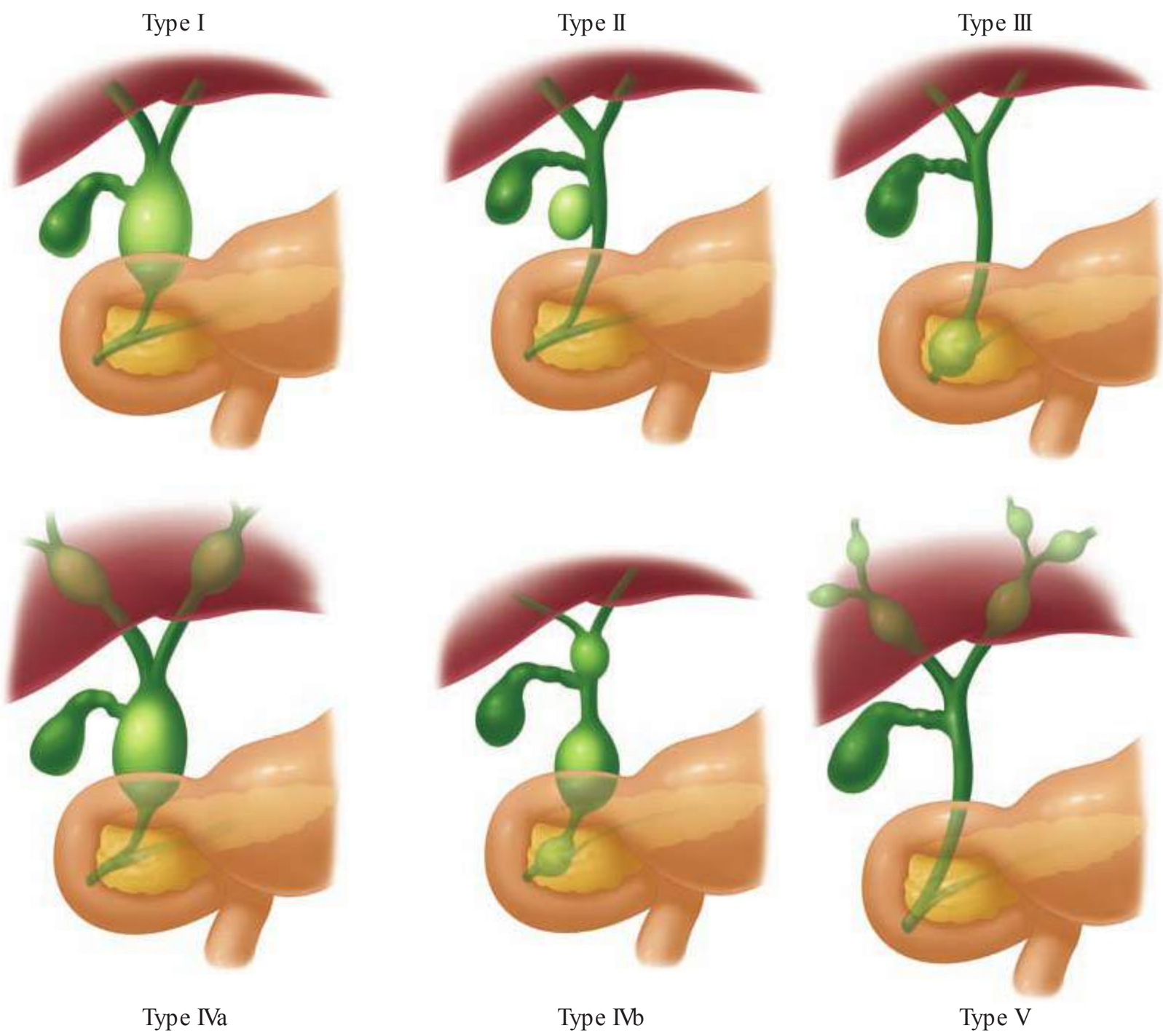


FiGUre 13-11. Todani classif cation of choledochal cysts. Type I, fusiform or cystic dilations of the extrahepatic biliary tree, is the most common type, making up > 50% of the choledochal cysts. Type II, saccular diverticulum of an extrahepatic bile duct. Rare, < 5% of choledochal cysts. Type III, bile duct dilatation within the duodenal wall (choledochoceles), makes up about 5% of choledochal cysts. Types IVa and IVb, multiple cysts, make up 5–10% of choledochal cysts. Type IVa affects both extrahepatic and intrahepatic bile ducts, whereas type IVb cysts affect the extrahepatic bile ducts only. Type V, intrahepatic biliary cysts, is very rare and makes up 1% of choledochal cysts.
(Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Figure 32-22.)

Treatment

- If patients present with cholangitis—Initial treatment with NPO, IV fluids, and broad spectrum antibiotics, then a def nitive surgical treatment.
- These cysts carry a risk of malignancy to develop cholangiocarcinoma.
- The surgical treatment is based on the type of the choledochal cyst.
- Type 1—Extrahepatic bile duct resection with the cyst, cholecystectomy with Roux-en-Y hepaticojejunostomy.
- Type 2—Simple cyst excision is an option, but might need bile duct resection with hepaticojejunostomy depending on degree of involvement.
- Type 3—Sphinterotomy or endoscopic resection.
- Type 4—Depending on the degree of involvement—extrahepatic bile duct resection with hepaticojejuostomy could be an option.
- Type 5—Liver transplant in ideal candidates. If not medical therapy to control symptoms.

CholangioCarCinoma

Def nition

An uncommon tumor that may occur anywhere along the intrahepatic or extrahepatic biliary tree but is most commonly located at the bifurcation of the right and left hepatic ducts (60–80% of cases). Nearly all are adenocarcinomas.

WARD TIP

A cholangiocarcinoma that arises at the junction of the right and left hepatic ducts is called a **Klatskin tumor.**

incidence

- Increases with age with peak at 55–65 years; 1/100,000 people per year.
- No sex predilection.

r isk Factors

Choledochal cyst, ulcerative colitis, sclerosing cholangitis, liver flukes, toxins, contrast dye.

Signs and Symptoms

- Jaundice
- Clay-colored stools
- Dark urine
- Pruritus
- Pain
- Malaise
- Weight loss

Diagnosis

- Ultrasound: Shows bile duct dilation.
- CT: Identif es tumors located near the hilum of the liver.
- MRI/MRCP: Useful in localizing the extent and site of obstruction.
- Biopsy via ERCP/PTC under ultrasound guidance.
- Classif cation of hilar cholangiocarcinoma: Bismuth classif cation (Figure 13-12)
- Type I—involves the common hepatic duct, spares the bifurcation
- Type II—involves the common hepatic duct and the bifurcation
- Type IIIa—tumor extends to the right branch of the hepatic duct
- Type IIIb—tumor extends to the left branch of the hepatic duct
- Type IV—tumor extends to both right and left branches

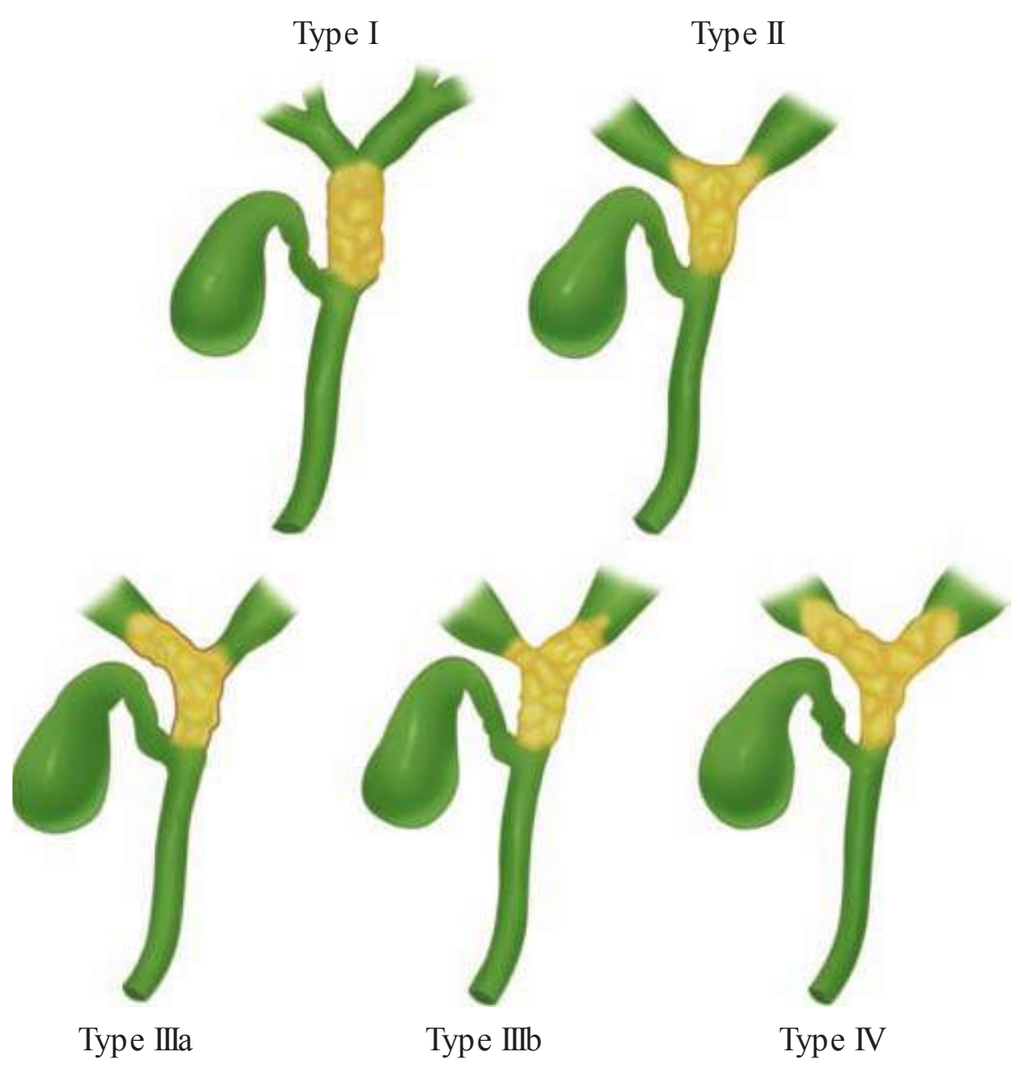


FiGUre 13-12. bismuth classif cation of hilar cholangiocarcinoma. Bismuth-Corlette classif cation of bile duct tumors. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Figure 32-26.)

Treatment

- Varies depending on location of the tumor.
- Proximal tumors: Resect with a Roux-en-Y hepaticojejunostomy.
- Distal tumors: Whipple procedure.
- If both hepatic ducts or the main trunk of the portal vein are extensively involved, the tumor may be unresectable.

prognosis

Five-year survival rate is 15–20%.

HIGH YIELD FACTS IN

The Pancreas

Embryology 216

Anatomy 216

Blood Supply and Innervation 216

Physiology 217

Exocrine 217

Endocrine 217

Acute Pancreatitis 218

definition 218

Etiology 218

Signs and Symptoms 218

diagnosis 218

differential diagnosis—pathology of structures in the Epigastrium, ruQ, lower mediastinum, and chest 219

treatment 219

prognosis 220

Chronic Pancreatitis 220

definition 220

Etiology 221

Signs and Symptoms 221

diagnosis 221

differential diagnosis 221

indications for Surgery 222

treatment 222

Other Complications of Pancreatitis 222

Pancreatic Adenocarcinoma 222

Etiology 222

risk factors 222

Signs and Symptoms 223

diagnosis 223

treatment 223

prognosis 223

Cystadenocarcinoma 224

Cystadenoma 224

Endocrine Tumors (Islet Cell Tumors) 224

insulinoma 224

gastrinoma 225

Vipoma 226

glucagonoma 226

Somatostatinoma 227

Pancreatic Trauma 227

Pancreas Transplantation 227

OR TIP

Resection of head of pancreas also requires resection of duodenum due to their shared blood supply (gastroduodenal artery).

Embryology

- During the fourth week of gestation, the pancreas begins development from the duodenal endoderm.
- Two buds form, which rotate and fuse by eighth week.
  - Ventral pancreatic bud: Uncinate process and part of the head.
  - Dorsal pancreatic bud: Remaining part of the head, neck, body, and tail.
- **Heterotopic pancreas:** Pancreatic tissue in an abnormal location such as the stomach, duodenum, or Meckel's diverticulum.
- **Pancreas divisum:** Due to a failure of the ventral and dorsal ducts to fuse, the majority of pancreatic drainage is accomplished via the accessory papilla and duct of Santorini. This is the most common congenital anomaly of the pancreas (5% of population) but is usually asymptomatic. Rarely, however, chronic pain and recurrent pancreatitis may result from inadequate drainage, requiring papillotomy—ERCP or surgical.
- **Annular pancreas:** Usually presents in infancy with duodenal obstruction (postprandial vomiting). Caused by malrotation of the ventral pancreas leading to a ring of pancreatic tissue around the second portion of the duodenum. Pancreatitis and peptic ulcers may also result.
- **Treatment:** Duodenojejunostomy.

Anatomy

- Location: Retroperitoneal (posterior to stomach, transverse mesocolon, and lesser omentum) at the level of the body of L2.
- Head: Includes uncinate process and abuts the second part of the duodenum.
- Neck: Portion overlying the superior mesenteric vein.
- Body: Lies to the left of the neck, forms posterior floor of lesser sac (omental bursa).
- Tail: Enters splenorenal ligament, adjacent to splenic hilum; susceptible to injury during splenectomy.
- Ducts: The duct of Wirsung is the main duct; runs entire length of pancreas. It joins the common bile duct and empties into the second part of the duodenum at the ampulla of Vater. The duct of Santorini (small duct) is an accessory duct often joining the duodenum more proximally than the ampulla of Vater.
- Sphincter of Oddi: Smooth muscle around the ampulla of Vater.

Blood Supply and Innervation

See Figure 14-1.

- Head:
  - **Anterior and posterior superior pancreaticoduodenal arteries**—branches of the gastroduodenal artery.
  - **Anterior and posterior inferior pancreaticoduodenal arteries**—branches of the superior mesenteric artery.
- Neck, body, and tail:
  - **Splenic artery and branches (dorsal pancreatic artery).**
- Sympathetics: Pain sensation is provided by the celiac plexus (via the splanchnic nerves).
- Parasympathetics: Islets, acini, and ducts are innervated by branches of the vagus nerve.

WARD TIP

Celiac plexus block can be done for pain control in pancreatic disease.

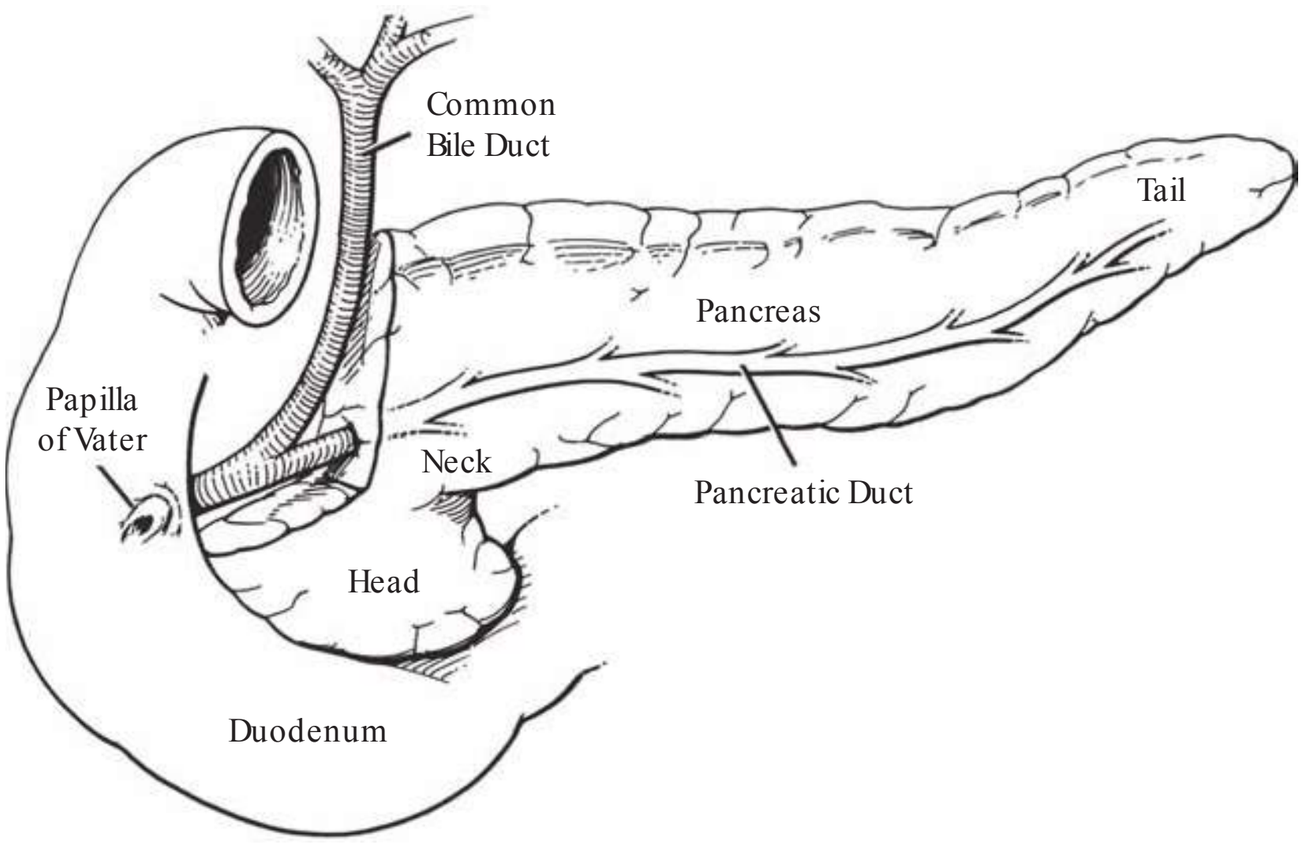


FIGURE 14-1. **The pancreas and its relationships.**

Physiology

Exocrine

Secretion of 1–2 L/day of clear, isosmotic, alkaline (pH 7.0–8.3) fluid containing digestive enzymes.

- **Acinar cells:** Secrete enzymes (e.g., chymotrypsin, trypsin, carboxypeptidase, amylase, lipase). These enzymes are secreted as inactive zymogen granules until they are activated intraluminally by enterokinase in the duodenum.
- **Centroacinar and ductal cells:** Secrete water and electrolytes (e.g., Na^+, K^+, HCO_3^-, Cl^-) in response to secretin stimulation.
- Phases:
  1. Cephalic phase: Sight and smell of food activates vagal fibers. Gastric acid production → duodenal acidification → secretin release → pancreatic HCO_3^- release.
  2. Gastric phase: Antral distention and ingested protein cause release of gastrin → gastric acid secretion → duodenal acidification → secretin release → pancreatic HCO_3^- release.
  3. Intestinal phase: Duododenal acid and bile stimulate secretin. Ingested fat and protein in the duodenum stimulate release of CCK → acinar cells release pancreatic enzymes.

EXAM TIP

Secretin is the most potent endogenous stimulant of bicarbonate secretion.

EXAM TIP

The exocrine pancreas makes up 85% of pancreatic volume; the endocrine pancreas accounts for only 2%, with the rest composed of extracellular matrix and vessels or ducts.

Endocrine

Islets of Langerhans make up 2% of pancreas by weight:

- Insulin: From **beta** cells in islets of Langerhans (glucose absorption and storage).
- Glucagon: From islet **alpha** cells (glycogenolysis and release of glucose).
- Somatostatin: From islet **delta** cells (generally causes inhibitory functions of gastrointestinal tract).

EXAM TIP

Gluc**a**gon = **a**lpha cells

EXAM TIP

Somatostatin may be used clinically to:
- Treat symptoms of neuroendocrine tumors (islet cell, carcinoid, gastrinoma, VIPoma, and acromegaly).
- Convert high-output fistulae to low-output fistulae (because of its antimotility and antisecretory effects).

EXAM TIP

Causes of pancreatitis:
Posterior perforation of peptic ulcer
alcohol
neoplasm
cholelithiasis (biliary disease)
renal disease (end stage)
eRCP
anorexia (malnutrition)
Trauma
Infections
Toxins (drugs)
Incineration (burns)
surgery/**s**corpion bite

EXAM TIP

Beware of **MeDVIPs**, which may cause drug-induced pancreatitis.
Methyldopa/**M**etronidazole
estrogen
Didanosine
Valproate
Isoniazid
Pentamidine
sulfonamides

Acute Pancreatitis

Definition

Inflammation of the pancreas due to parenchymal autodigestion by proteolytic enzymes.

Etiology

Most common etiologies in the United States:

1. Alcohol abuse (40–50%)
2. Gallstones (40%)
3. Idiopathic (10%)

Other causes of acute pancreatitis:
- Hyperlipidemia
- Hypercalcemia: Secondary to hyperparathyroidism
- Trauma
- Postop and post-ERCP (endoscopic retrograde cholangiopancreatography)
- Pancreatic duct obstruction (e.g., tumor, pancreatic divisum)
- Vasculitis
- Scorpion venom
- Viral infection (e.g., mumps, coxsackie B, cytomegalovirus)
- Drugs (e.g., isoniazid, glucocorticoids, cimetidine)

Signs and Symptoms

- Severe, constant epigastric pain **radiating to the back (because pancreas is a retroperitoneal organ and is innervated by the celiac plexus).** Pain may be improved by sitting forward or standing.
- Nausea, vomiting.
- Physical exam:
  - Low-grade fever, tachypnea, tachycardia, upper abdominal tenderness with guarding but no rebound. Bowel sounds may be absent due to adynamic ileus. Signs of hypovolemic shock may also be present due to massive retroperitoneal fluid sequestration, dehydration, and systemic inflammatory response syndrome.
  - Cullen's sign (bluish discoloration of periumbilicus), Grey-Turner's sign (bluish discoloration of flank), and Fox's sign (bluish discoloration of inguinal ligament) are indicative of severe, hemorrhagic pancreatitis.

Diagnosis

Laboratory Studies

- Elevated lipase: Only found in gastric and intestinal mucosa and liver, in addition to the pancreas, so is more specific for pancreatitis than amylase.
- Elevated amylase:
  - Also found in salivary glands, small bowel, ovaries, testes, and skeletal muscle, so is not a specific marker for pancreatitis.
  - Although amylase may be persistently elevated in renal insufficiency, a level three times the upper limit of normal is suggestive of pancreatitis.
  - Neither amylase nor lipase is a part of Ranson's criteria.
  - Ranson's criteria listed in Table 14-1.

TABLE 14-1. Ranson's Criteria (Predicts Risk of Mortality in Pancreatitis)

On Admission	After 48 Hours
Age > 55	Base deficit > 4
Blood sugar > 200	Increase in blood urea nitrogen (BUN) > 5
Serum aspartate aminotransferase (AST) < 250	Fluid deficit > 6 L
Lactic dehydrogenase (LDH) > 350	Calcium < 8
White blood count (WBC) > 16,000	Drop in hematocrit > 10%
	PO_2 < 60 mmHg

Number Of Risk Factors	Mortality
< 3	1%
3 or 4	16%
5 or 6	40%
> 6	70–100%

Abdominal Imaging

- Abdominal x-ray: Sentinel loop sign and colon cutoff sign.
- Ultrasound: May demonstrate pseudocysts, phlegmon, abscesses, or cholelithiasis.
- Computed tomography (CT) scan: Diagnostic test of choice (90% sensitive and 100% specific).
- Demonstrates
  - the degree of pancreatitis—edematous gland to severe stranding around the pancreas and fluid collections. Helpful in grading of pancreatitis—Balthazar grading system.
  - complications, phlegmon, abscesses, pancreatic necrosis, or pseudocyst formation (see Figure 14-2).

Differential Diagnosis—Pathology of Structures in the Epigastrium, RUQ, Lower Mediastinum, and Chest

- Acute cholecystitis
- Gastritis/peptic ulcer disease
- Perforated esophagus/peptic ulcer
- Myocardial infarction
- Ruptured abdominal aortic aneurysm
- Acute mesenteric ischemia
- Pneumonia

Treatment

- Aggressive hydration with electrolyte monitoring to maintain adequate intravascular volume, preferably in intensive care unit (ICU) setting.

WARD TIP

Cullen's sign: Think of cULLen and UmbiLicus.
Grey-Turner's sign: Think of **TUr n** ing on your side/flank.

WARD TIP

High amylase levels are also seen in intestinal disease, perforated ulcer, ruptured ectopic pregnancy, salpingitis, salivary gland disorders, renal failure, and diabetic ketoacidosis.

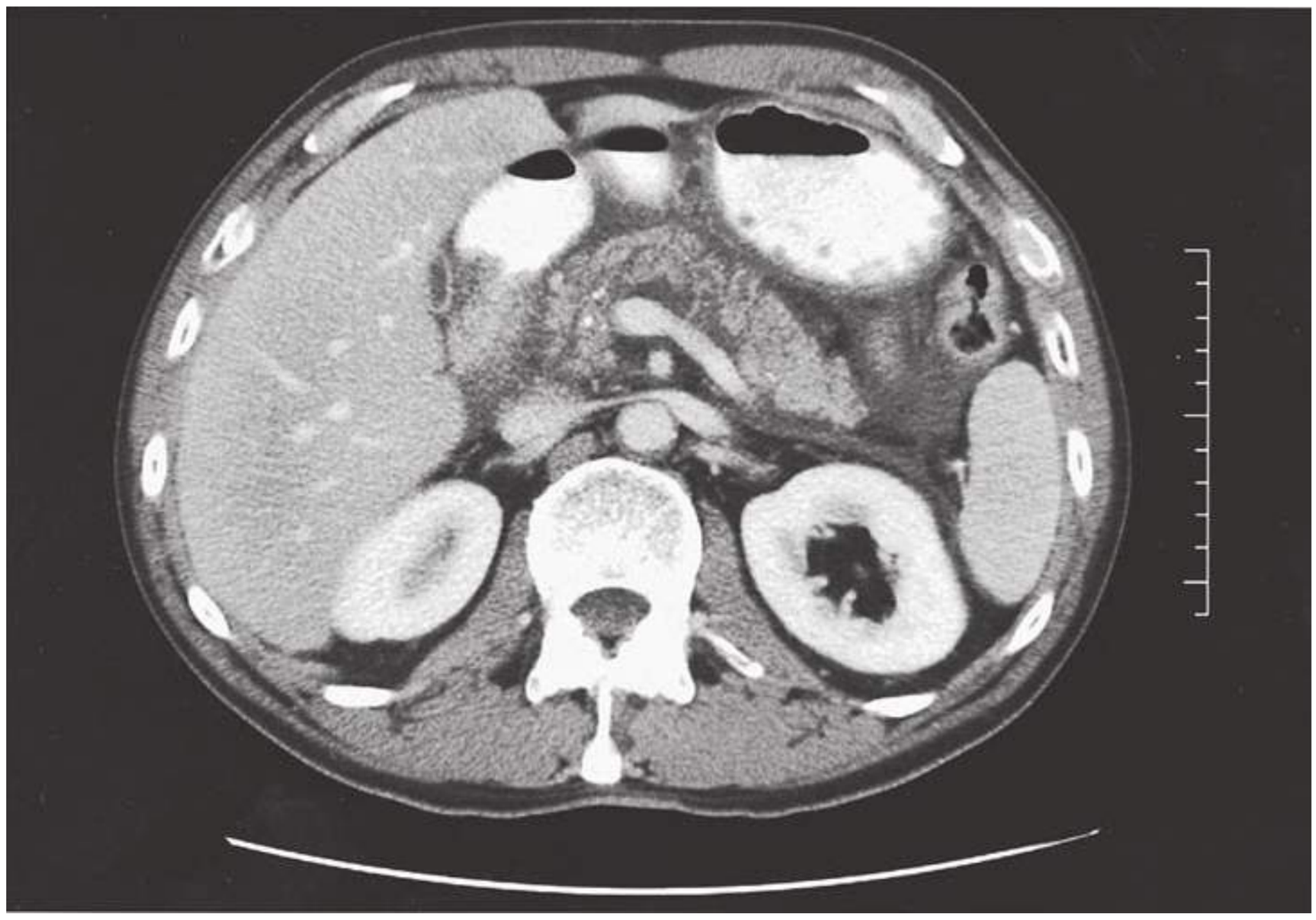


FIGURE 14-2. Abdominal CT demonstrating stranding in the peripancreatic region, consistent with acute pancreatitis.

- Nasogastric tube: For severe disease with vomiting.
- Antibiotics: If infection identified especially in necrotizing pancreatitis (CT guided aspiration).
- NPO with nutritional support via post pyloric feeding (enteral feeding is proven to beneficial compared to TPN) or total parenteral nutrition (TPN).
- Avoid use of morphine due to possible spasm of sphincter of Oddi. Use meperidine (Demerol) instead for pain management.
- Surgery indicated for:
  - Complications of pancreatitis—infected necrosis, hemorrhage, pseudocysts.
  - Correction of associated biliary tract disease: Gallstone pancreatitis should be treated with early interval cholecystectomy only after acute pancreatic inflammation has resolved to prevent further attacks of pancreatitis which can be of higher severity. Acutely, ERCP with endoscopic sphincterotomy may be used to relieve biliary obstruction.

EXAM TIP

A sentinel loop is distention and/or air-fluid levels near a site of inflammation. In pancreatitis, it is secondary to pancreatitis-associated ileus.

Prognosis

See Table 14-1 for Ranson's criteria.

Chronic Pancreatitis

Definition

Chronic inflammation or recurrent acute pancreatitis causes irreversible parenchymal fibrosis, destruction, and calcification, leading to loss of **endocrine** and **exocrine** function.

Etiology

- Most commonly alcohol abuse (70%).
- Idiopathic (20%).
- Other (10%): Hyperparathyroidism, hypertriglyceridemia, congenital pancreatic anomalies, hereditary, obstruction.

Signs and Symptoms

- Recurrent or constant epigastric and/or back pain, bloating, abdominal cramps.
- Malabsorption/malnutrition (exocrine dysfunction).
- Steatorrhea (exocrine dysfunction)—fat-soluble vitamin deficiency.
- Type 1 diabetes mellitus (endocrine dysfunction).
- Polyuria.

diagnosis

- History.
- Fecal fat analysis—both quantitative and qualitative (Sudan stain) stool for elastase levels.
- X-ray (kidneys, ureters, bladder): **Pancreatic calcifications.**
- ERCP or MRCP: **Chain-of-lakes** pattern—ductal irregularities with dilation and stenosis.
- CT: **Pseudocysts** (see Figure 14-3) (use ultrasound for follow-up of pseudocysts). **Gland enlargement/atrophy, calcifications, masses** also seen on CT.

differential diagnosis

- Peptic ulcer disease.
- Pancreatic cancer.
- Biliary tract disease (cholecystitis).

WARD TIP

Gallstones are not a common cause of chronic pancreatitis.

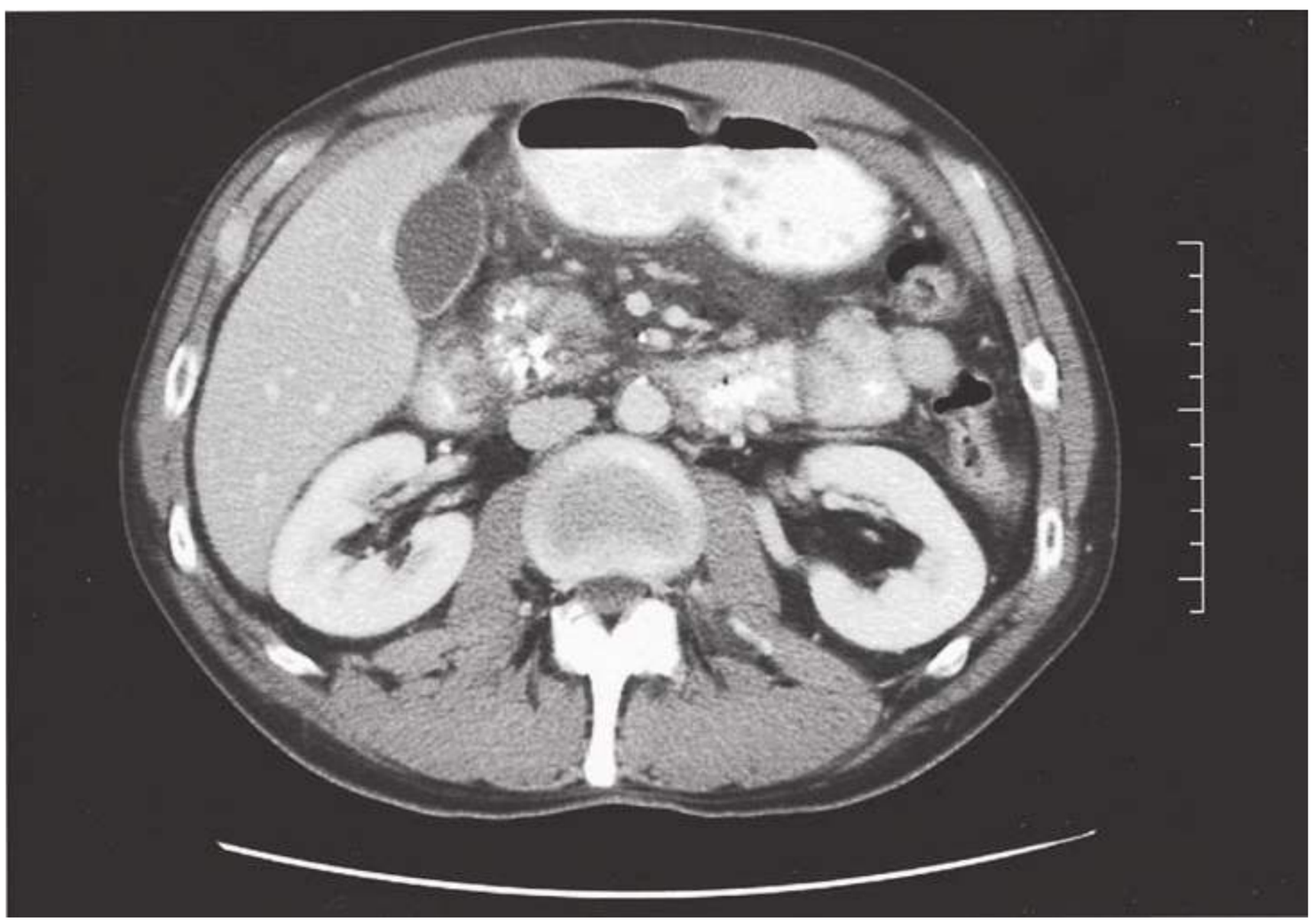


FIGURE 14-3. Abdominal CT demonstrating calcification involving the entire head of the pancreas, consistent with chronic pancreatitis.

Indications for Surgery

- Persistent pain
- Gastrointestinal or biliary obstruction
- Pseudocyst infection, hemorrhage, or rupture
- Enlarging pseudocysts

Treatment

- **Nonoperative management:** Includes control of abdominal pain, endocrine and exocrine insufficiency (insulin and pancreatic enzyme therapy).
- **Operative management** (can do preop MRCP or ERCP to evaluate anatomy):
- Pain relief: Celiac plexus block.
- Ampullary procedures: ERCP with endoscopic sphincterotomy.
- Ductal decompression/drainage procedures:
  - Puestow procedure (longitudinal pancreaticojejunostomy) for segmental ductal dilation.
  - Duval procedure (retrograde drainage with distal resection and end-to-end pancreaticojejunostomy).
- Resective procedures (resection of portions of pancreas):
  - Frey procedure (longitudinal pancreaticojejunostomy with partial resection of the pancreatic head).
  - Whipple procedure (pancreaticoduodenectomy with hepaticojejunostomy, pancreaticojejunostomy, and gastrojejunostomy).
  - Beger procedure—coring out the pancreatic parenchyma in the head of the pancreas (if major involvement is the pancreatic head only)
  - Near-total or total pancreatectomy
- Pseudocysts: Nonepithelialized, encapsulated pancreatic fluid collections. Up to 30% of peripancreatic fluid collections resolve on their own with bowel rest (TPN and NPO). If after 6 weeks they have not resolved they will tend to have a mature wall and are termed pseudocysts. Pseudocysts larger than 6 cm in size rarely resolve spontaneously and internal drainage of the mature cyst is indicated via cyst gastrostomy or Roux-en-Y cyst jejunostomy.

WARD TIP

Pancreatic calcifications and stones are associated with chronic pancreatitis.

Other Complications of Pancreatitis

- Pancreatic abscess
- Pancreatic fistulae
- Hemorrhagic pancreatitis
- Pancreatic cancer

Pancreatic Adenocarcinoma

Etiology

- Originates in the exocrine pancreas (ductal cells).
- Two thirds occur in the head of the pancreas.

Risk factors

- Male gender (2:1 male-to-female).
- African-American heritage (2:1 Black-to-White).

WARD TIP

Tumors of the pancreatic tail usually present at a late stage because they are asymptomatic during growth. Tumors in the head or uncinate process of the pancreas, however, often present earlier because they cause **biliary obstruction.**

- Tobacco use (2× increased risk).
- Diabetes, alcohol abuse, chronic pancreatitis, and increased age are also associated with increased risk.

Signs and Symptoms

- Constitutional symptoms—weight loss and loss of appetite.
- Painless jaundice (due to ampullary obstruction).
- New onset diabetes and steatorrhea (endocrine and exocrine insuffciency).
- Epigastric pain radiating to the back—usually due to underlying chronic pancreatitis.
- Migratory thrombophlebitis: Trousseau's syndrome is seen especially in tumors of the body or tail.

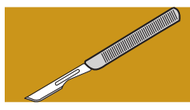


WARD TIP

courvoisier's sign: Jaundice with a palpable nontender gallbladder.

diagnosis

- Elevated carcinoembryonic antigen (CEA) or CA 19-9 (tumor markers).
- Abdominal CT scan with IV contrast is study of choice. Classic double duct sign—dilated CBD and pancreatic duct, with a hypodense mass in the head of the pancreas.
- MRI/MRCP is also useful.
- Percutaneous transhepatic cholangiography (PTC) and ERCP (useful in periampullary lesions.
- Angiography may also be useful.



OR TIP

Whipple procedure: Removal of gallbladder, common bile duct (CBD), antrum of stomach, duodenum, proximal jejunum, and head of pancreas (en bloc); Reconstruction with pancreaticojejunostomy, hepaticojejunostomy, and gastrojejunostomy.

treatment

- Tumors of the head: The only chance for a cure is the Whipple procedure (pancreaticoduodenectomy), and most tumors are **not** resectable at the time of diagnosis.
- Tumors of the body/tail: Distal "near-total" pancreatectomy.
- If unresectable (due to liver/peritoneal metastases, nodal metastases beyond the zone of resection, or tumor invasion of the superior mesenteric artery), palliative procedure considered:
  - **Relieve biliary obstruction—PTC, ERCP with biliary stenting, cholecystojejunostomy, or choledochojejunostomy**
  - **Relieve duodenal obstruction—gastrojejunostomy—bypass procedure.**
- Double bypass—gastric and biliary bypass procedure.
- **Splanchnic nerve block (pain control) or neurolysis with absolute alcohol.**
- Postoperative chemoradiation therapy typically includes 5-fluorouracil (5-FU) and external-beam radiation.
- If tumor is borderline resectable invading the adjacent superior mesenteric vein neoadjuvant chemotherapy is an option to make them resectable.

prognosis

- The mean survival for patients with unresectable disease is 4–6 months, with a 5-year survival rate of < 3%.
- The median survival for patients who undergo successful resection is approximately 12–19 months, with a 5-year survival rate of 15–20%.

Cystadenocarcinoma

- Commonly seen in females age 40–60 years.
- In body and tail.
- Less than 2% of all pancreatic exocrine tumors.
- Prognosis better than adenocarcinoma.
- **Treatment:** Surgical resection—distal/total pancreatectomy.

Cystadenoma

- Older and middle-aged women
- Two types:
  - Serous: Benign
  - Mucinous: Generally benign but has potential to be malignant
- **Treatment:** Surgical resection

WARD TIP

Pancreatic cystadenoma: **M**ucinous = **M**alignant potential

Endocrine Tumors (Islet Cell Tumors)

Insulinoma

Definition

Beta cell neoplasm with overproduction of insulin.

Epidemiology

- Most common islet cell tumor.
- Ninety percent are benign.
- Most are solitary lesions with even distribution in the head, body, and tail of the pancreas.
- If associated with multiple endocrine neoplasia (MEN) I syndrome (< 10% of cases), then multiple insulinomas may be present.

EXAM TIP

Typical scenario: A 30-year-old male complains of feeling faint and confused most notably after he exercises. His symptoms improve after he has a soft drink. Think: Insulinoma—check fasting serum insulin level.

Signs and Symptoms

Most symptoms related to hypoglycemia triggered catecholamine release.

Differential Diagnosis

Surreptitious insulin administration.

Diagnosis

- Fasting serum insulin level > 10 μU/mL (normal: < 6 μU/mL).
- Fasting insulin-to-glucose ratio > 0.3.
- Proinsulin or C-peptide levels should be measured to rule out surreptitious exogenous insulin administration. Abdominal CT scan or MRI—hypervascular nodules.
- Selective mesenteric angiography can be considered to localize.

Treatment

- Surgical enucleation or resection is usually curative (90% of patients).
- Diazoxide can improve hypoglycemic symptoms by inhibiting pancreatic insulin release.

WARD TIP

Insulinoma is characterized by **Whipple's triad:**

1. Symptoms of hypoglycemia with fasting
2. Fasting glucose < 50 mg/dL
3. Relief of symptoms after eating

gastrinoma

Definition

- Neoplasm associated with overproduction of gastrin.
- Also known as Zollinger–Ellison syndrome.

epidemiology

- Second most frequent islet cell tumor.
- Ninety percent are located in the "gastrinoma triangle" (Figure 14-4) bordered by:
  - Junction of second and third part of the duodenum.
  - The cystic duct.
  - The superior mesenteric artery under the neck of the pancreas.
- Small, slow-growing, multiple, 60% malignant.

EXAM TIP

Twenty-five percent of gastrinomas are associated with MEN-I.

signs and symptoms

- Signs of peptic ulcer disease (especially in patients with recurrent or unusually located ulcers).
- Epigastric pain most prominent after eating.
- Profuse watery diarrhea.

Diagnosis

- Fasting serum gastrin level > 500 pg/mL (normal: < 100 pg/mL).
- Secretin stimulation test will cause a paradoxical increase in gastrin in patients with Zollinger–Ellison syndrome (double fasting level or increase of 200 pg/dL over the fasting level).
- Ulcers in unusual locations (e.g., third part of duodenum or jejunum) is highly suggestive.
- Octreotide scan to localize tumor.

EXAM TIP

Typical scenario: A 40-year-old male complains of chronic epigastric pain shortly following meals and notices needing increasing doses of his anti-ulcer medication. Think: Gastrinoma.

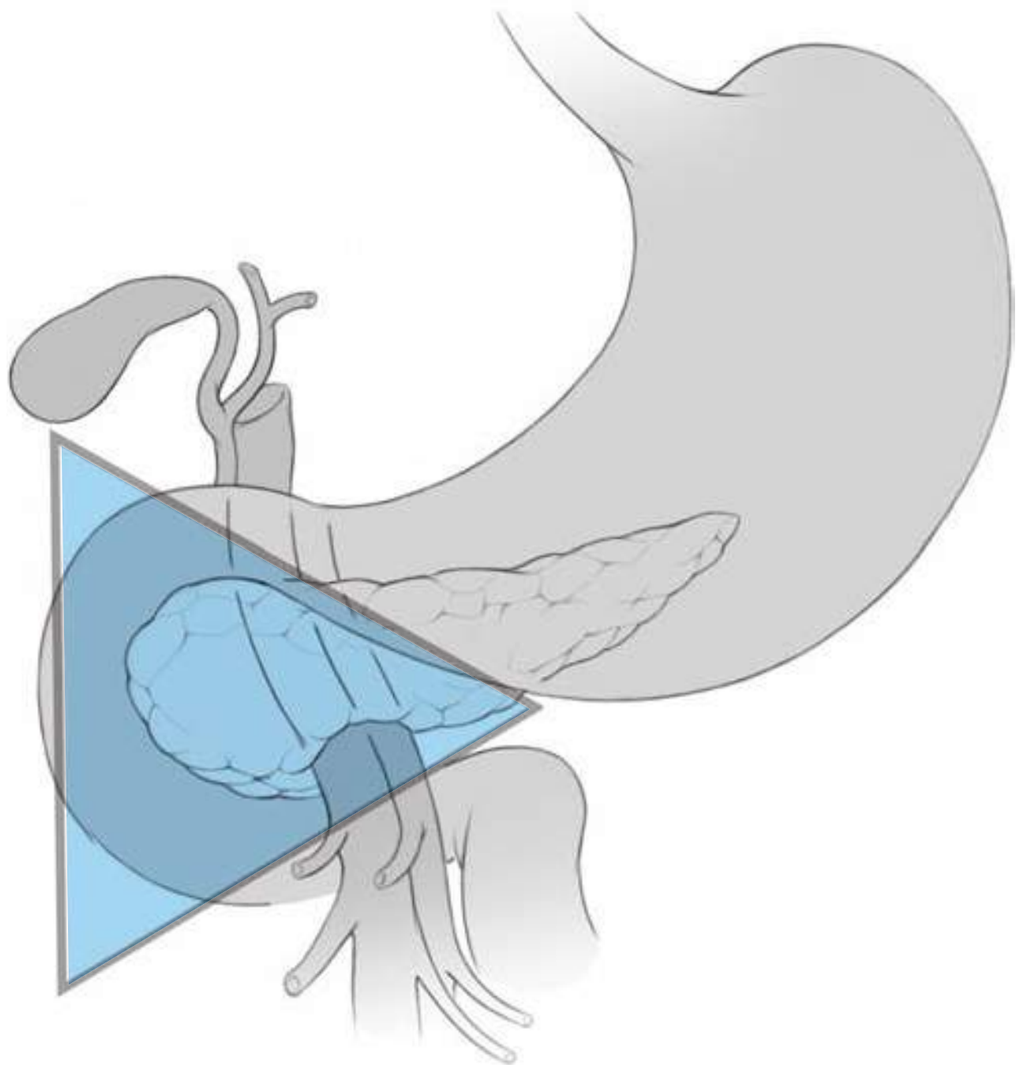


FIGURE 14-4. Gastrinoma triangle—the anatomic triangle in which approximately 90% of gastrinomas are found.

Treatment

- Proton pump inhibitor to alleviate symptoms.
- Surgical resection (curative or debulking).
- Chemotherapy.

Vipoma

Definition

Overproduction of vasoactive intestinal peptide (VIP).

epidemiology

- Also known as Verner–Morrison syndrome or **WDHA** syndrome (**W**atery **D**iarrhea, **H**ypokalemia, **A**chlorhydria).
- Most are malignant; majority have metastasized to lymph nodes and the liver at time of diagnosis.
- Ten percent are extrapancreatic.

signs and symptoms

- Severe watery diarrhea.
- Signs of hypokalemia—palpitations/arrhythmias, muscle fasiculations/tetany, paresthesias.

EXAM TIP

Typical scenario: A 57-year-old male presents with a history of severe watery diarrhea characterized by hypokalemia and achlorhydria. His most recent bout required fluid resuscitation in the intensive care unit (ICU). Think: VIPoma.

Diagnosis

Fasting serum VIP level > 800 pg/mL (normal: < 200 pg/mL) with exclusion of other causes of diarrhea.

Treatment

- Surgical resection, chemotherapy.
- Octreotide (somatostatin analogue)—diarrhea control.

glucagonoma

Definition

Rare alpha cell neoplasm resulting in overproduction of glucagon.

epidemiology

Most are malignant, large primary tumors that have usually metastasized to lymph nodes and liver at the time of diagnosis.

signs and symptoms

- Mild diabetes (hyperglycemia).
- Anemia.
- Mucositis.
- Weight loss due to low amino acid levels.
- Necrolytic migratory erythema—a red psoriatic-like rash with serpiginous borders over trunk and lower limbs.

EXAM TIP

Necrolytic migratory erythema is the skin condition associated with glucagonoma.

Diagnosis

- Fasting serum glucagon level > 1,000 pg/mL (normal: < 200 pg/mL).
- Skin biopsy to confirm presence of necrolytic migratory erythema.

Treatment

- Surgery and chemotherapy.
- Octreotide to inhibit the release of glucagon.

Somatostatinoma

- Very rare tumor.
- Malignant.
- Treated by surgery and chemotherapy.

Pancreatic Trauma

See Trauma chapter.

Pancreas Transplantation

See Transplant chapter.

HIGH-YIELD FACTS IN

The endocrine System

Hormones 230
- Definition 230
- Classes 230
- Mechanisms of action 230

Multiple Endocrine Neoplasia (MEN) 233
- General 233
- Men i (Wermer's syndrome) 233
- Men iia (Sipple's syndrome) 233
- Men iiB 234

Thyroid 234
- General 234
- Development 234
- anatomy 234
- vasculature 235
- innervation 236
- hormones 236
- assessment of function 237
- Congenital anomalies 237
- hyperthyroidism 237
- hypothyroidism 239
- thyroiditis 240
- Workup of a Mass 240
- thyroid Cancer 241

Parathyroid 243
- Development 243
- anatomy 243
- physiology/Calcium homeostasis 243
- pth 243
- Calcitonin 244
- hyperparathyroidism 244
- hypoparathyroidism 245
- parathyroid Cancer 246

Adrenal Gland 246
- anatomy 246
- physiology 246
- hyperplasia 247
- adenoma 247
- adrenal cortical carcinoma 247
- Cushing's syndrome 248
- addison's Disease 250
- hyperaldosteronism 251
- hypoaldosteronism 252

Medullary Tumors 252
- neuroblastoma 252
- pheochromocytoma 253

Pituitary 254
- anatomy 254
- adenoma 254
- Sheehan's syndrome 255
- posterior pituitary Disorders 256

Hormones

Definition

A chemical substance secreted into the bloodstream by cells in one part of the body that acts on distant organs or tissues.

Classes

- Steroid:
  - Adrenal cortical: Cortisol, aldosterone.
  - Ovarian, testicular, and placental hormones (not discussed here).
- Tyrosine-derived:
  - Thyroid: Triiodothyronine (T_3), thyroxine (T_4).
  - Adrenal medulla: Epinephrine, norepinephrine.
- Protein/peptide:
  - Pituitary:
    - Anterior: Growth hormone (GH), adrenocorticotropic hormone (ACTH), thyroxine-stimulating hormone (TSH), follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin (PRL).
    - Posterior: Antidiuretic hormone (ADH), oxytocin.
  - Parathyroid: Parathyroid hormone.
  - Pancreas: Insulin, glucagon, somatostatin.

WARD TIP

All **protein** hormones are produced in locations starting with **P:**
Pituitary
Parathyroid
Pancreas

Mechanisms of Action

See Table 15-1.

TABLE 15-1. Summary of Endocrine Hormones and Their Functions

Endocrine Organ	Hormone	Functions	Stimulated By	Inhibited By
Anterior pituitary	Growth hormone (GH)	▪ Opposes insulin ▪ Stimulates amino acid uptake ▪ Stimulates release of fatty acid from storage sites ▪ Mediates immunoglobulin F (IGF) synthesis ▪ Stimulates growth of nearly all tissues	▪ Growth hormone–releasing hormone (GHRH) ▪ Hypoglycemia ▪ Arginine ▪ Exercise ▪ l-dopa ▪ Clonidine ▪ Propranolol	Somatostatin
	Adrenocorticotropic hormone (ACTH)	▪ Stimulates secretion of adrenocortical hormones	▪ Corticotropin-releasing hormone (CRH) ▪ Stress	Cortisol (negative feedback)
	Thyroid-stimulating hormone (TSH)	▪ Regulates secretion of triiodothyronine (T_3) and thyronine (T_4)	▪ Thyrotropin-releasing hormone (TRH)	Negative feedback
	Follicle-stimulating hormone (FSH)	▪ Stimulates ovarian follicular growth (female) ▪ Stimulates spermatogenesis and testicular growth (male)	▪ Gonadotropin-releasing hormone (GnRH)	Negative feedback

(continues)

TABLE 15-1. Summary of Endocrine Hormones and Their Functions (continued)

Endocrine Organ	Hormone	Functions	Stimulated By	Inhibited By
Anterior pituitary (continued)	Luteinizing hormone (LH)	▪ Ovulation (female) ▪ Luteinization of follicle (female) ▪ Stimulates production of estrogen and progesterone (female) ▪ Promotes production of testosterone (male)	▪ GnRH	Negative feedback
	Prolactin (PRL)	▪ Facilitates breast development in preparation for milk production	▪ TRH ▪ Estrogen ▪ Stress ▪ Exercise	Bromocriptine
Posterior pituitary	Antidiuretic hormone (ADH, vasopressin)	▪ Promotes water absorption in collecting ducts of kidney ▪ Vasoconstriction of peripheral arterioles, increasing blood pressure	▪ Increased plasma osmolality ▪ Decreased plasma volume	
	Oxytocin	▪ Increases frequency and strength of uterine contractions ▪ Stimulates breast milk ejection	▪ Suckling ▪ Vaginal stimulation	
Thyroid	T_3, T_4	▪ Increase basal metabolic rate (BMR), oxygen consumption ▪ Increase protein synthesis, lipolysis, glycogenolysis, gluconeogenesis ▪ Increase heart rate and contractility ▪ Increase catecholamine sensitivity ▪ Stimulate release of steroid hormones ▪ Stimulate erythropoiesis and 2,3-diphosphoglycerate (DPG) production ▪ Increase bone turnover	TSH	Negative feedback
	Calcitonin	▪ Increases serum calcium (by inhibiting osteoclasts) ▪ Increases phosphate excretion	High serum calcium	Low serum calcium
Parathyroid	Parathyroid hormone (PTH)	▪ Kidney: Increases calcium resorption in proximal convoluted tubule ▪ Rapid ▪ Also increased excretion of sodium, potassium, phosphate, and bicarbonate ▪ Bone: Increases calcium mobilization ▪ Rapid phase: Equilibration with extracellular fluid (ECF) ▪ Slow phase: Enzyme activation (promoting bone resorption) ▪ GI tract (indirect): Increases absorption via vitamin D	Low serum calcium	High serum calcium

(continues)

TABLE 15-1. Summary of Endocrine Hormones and Their Functions (continued)

Endocrine Organ	Hormone	Functions	Stimulated By	Inhibited By
Pancreas	Insulin (B cell)	Effects on liver: ▪ Glycogenesis, glycolysis, synthesis of protein, triglycerides, cholesterol, very low-density lipoprotein (VLDL) ▪ Inhibits glycogenolysis, ketogenesis, gluconeogenesis Effects on muscle: ▪ Protein synthesis, glycogen synthesis Effects on fat: ▪ Promotes triglyceride storage	Hyperglycemia	Hypoglycemia
	Glucagon (A cell)	Metabolic effects: ▪ Glycogenolysis, gluconeogenesis, ketogenesis (liver); lipolysis (adipose tissue); insulin secretion Effects on gastrointestinal (GI) secretion: ▪ Inhibition of gastric acid and pancreatic exocrine secretion Effects on GI motility: ▪ Inhibition of peristalsis Cardiovascular effects: ▪ Increase in HR and force of contraction	Hypoglycemia	Hyperglycemia
	Somatostatin (D cell)	▪ Inhibition of gastric acid, pepsin, pancreatic exocrine secretion ▪ Inhibition of ion secretion ▪ Inhibition of motility ▪ Reduction of splanchnic blood flow ▪ Inhibition of insulin, glucagons, pancreatic polypeptide secretion		
	Pancreatic polypeptide (F cell)	▪ Function not known, but level rises after a meal (possible inhibition of pancreatic exocrine secretion)		
Adrenal cortex	Cortisol (zona fasciculata and reticularis)	▪ Stimulation of hepatic gluconeogenesis, inhibition of protein synthesis, increased protein catabolism, lipolysis, inhibition of peripheral glucose uptake ▪ Inhibition of fibroblast activity, inhibition of bone formation, reduction of GI calcium absorption ▪ Inhibition of leukocytes, decreased migration of inflammatory cells to site of injury, decreased production of mediators of inflammation	▪ ACTH circadian rhythm ▪ Stress	Negative feedback

(continues)

TABLE 15-1. Summary of endocrine hormones and Their Functions (continued)

Endocrine Organ	Hormone	Functions	Stimulated By	Inhibited By
	Androgens (zona fasciculata and reticularis)	▪ Dehydroepiandrosterone (DHEA), DHEA sulfate are converted to testosterone and dihydrotestosterone in the periphery ▪ Adrenal androgens make up < 5% of total testosterone production in the normal male		
	Aldosterone (zona glomerulosa)	▪ Stimulates renal tubular sodium absorption in exchange for potassium and hydrogen ▪ Net effect: Fluid reabsorption and intravascular volume expansion		
Adrenal medulla	Epinephrine and norepinephrine	▪ Increased oxygen consumption ▪ Increased heat production ▪ Stimulation of glycogenolysis, lipolysis, inhibition of insulin secretion	Stress Receptors α: Vasoconstriction β_1: Cardiac inotropic and chronotropic stimulation β_2: Noncardiac smooth muscle relaxation (vessels, uterus, bronchi)	

Multiple Endocrine Neoplasia (MEN)

General

Autosomal dominant genetic disorder.

Men i (Wer Mer's syndrome)

- Chromosomal deletion: 11q12–13.
- Parathyroid hyperplasia (90%).
- Pancreatic (and duodenal) islet cell tumors (50%).
- Pituitary adenomas (25%) (prolactinoma is most common).

Men iia (sipple's syndrome)

- RET oncogene mutation on chromosome 10q11.2; missense mutations on chromosome 1.
- Medullary thyroid carcinoma (100%)—20% of all medullary cancers are due to MEN.
- Pheochromocytoma (33%)—majority are bilateral.
- Parathyroid hyperplasia (50%).

EXAM TIP

MEN I tumors are the **3 Ps:**
Pituitary
Parathyroid
Pancreas

EXAM TIP

MEN IIA, 2Ps and M
- **P**heochromocytoma
- **P**arathyroid
- **m**edullary thyroid carcinoma

MEN IIB, 1P and 2Ms
- **P**heochromocytoma
- **m**edullary thyroid carcinoma
- **m**arfanoid habitus

WARD TIP

Persistence of the thyroid-pharynx connection may occur via a sinus or cyst, called a thyroglossal duct cyst. These cysts present as midline neck masses that move with swallowing and are usually seen in children or adolescents. They should be surgically excised because of the risk of infection.

Men iiB

- Mucosal neuroma may be earliest sign present (100%)—hypertrophied lips, thickened eyelids.
- Medullary thyroid carcinoma (85%).
- Pheochromocytoma (50%).
- Marfanoid habitus—skeletal abnormalities of spine (e.g., kyphosis), pectus excavatum.

Treatment

- Perform subtotal or total parathyroidectomy with autotransplantation for parathyroid hyperplasia (MEN I and MEN IIA).
- Perform total thyroidectomy with bilateral prophylactic central lymph node dissection for medullary thyroid cancer (MEN II).

Thyroid

General

The thyroid gland is responsible for the metabolic activity of the body. Dysfunction of the thyroid can result in hyper or hypo states of hormone production. Several different types of cancer can also form in the thyroid gland. These conditions may ultimately require surgical correction.

DevelopMent

- The thyroid develops at the base of the tongue between the first pair of pharyngeal pouches, in an area called the foramen cecum.
- The thyroid gland then descends down the midline to its final location overlying the thyroid cartilage, and develops into a bilobed organ with an isthmus between the two lobes.
- It remains connected to the f oor of the pharynx via the thyroglossal duct, which subsequently obliterates around the second month of gestation. However, the thyroglossal duct may fail to obliterate and form a thyroglossal cyst or fistula instead. These are most commonly seen in children and should be surgically excised.
- A pyramidal lobe can be seen in 50–80% of the population and represents a remnant of the distal thyroglossal tract. The pyramidal lobe extends superiorly from the median isthmus.

anat oMy

See Figure 15-1.

Gross Anatomy

- Two lobes, isthmus, pyramidal lobe (pyramidal lobe present in 50–80%).
- Suspended from larynx, attached to trachea (cricoid cartilage and tracheal rings).
- Weighs 20–25 g in adults.
- Relationships:
  - Anterior: Strap muscles (sternohyoid, sternothyroid, thyrohyoid, omohyoid).
  - Posterior: Trachea.
  - Posterolateral: Common carotid arteries, internal jugular veins, vagus nerves.
  - Parathyroid glands on posterior surface of thyroid, and may be within capsule.

EXAM TIP

Lymphatics ultimately drain to internal jugular nodes. Intraglandular lymphatics connect both lobes, explaining the relatively high frequency of multifocal tumors in the thyroid.

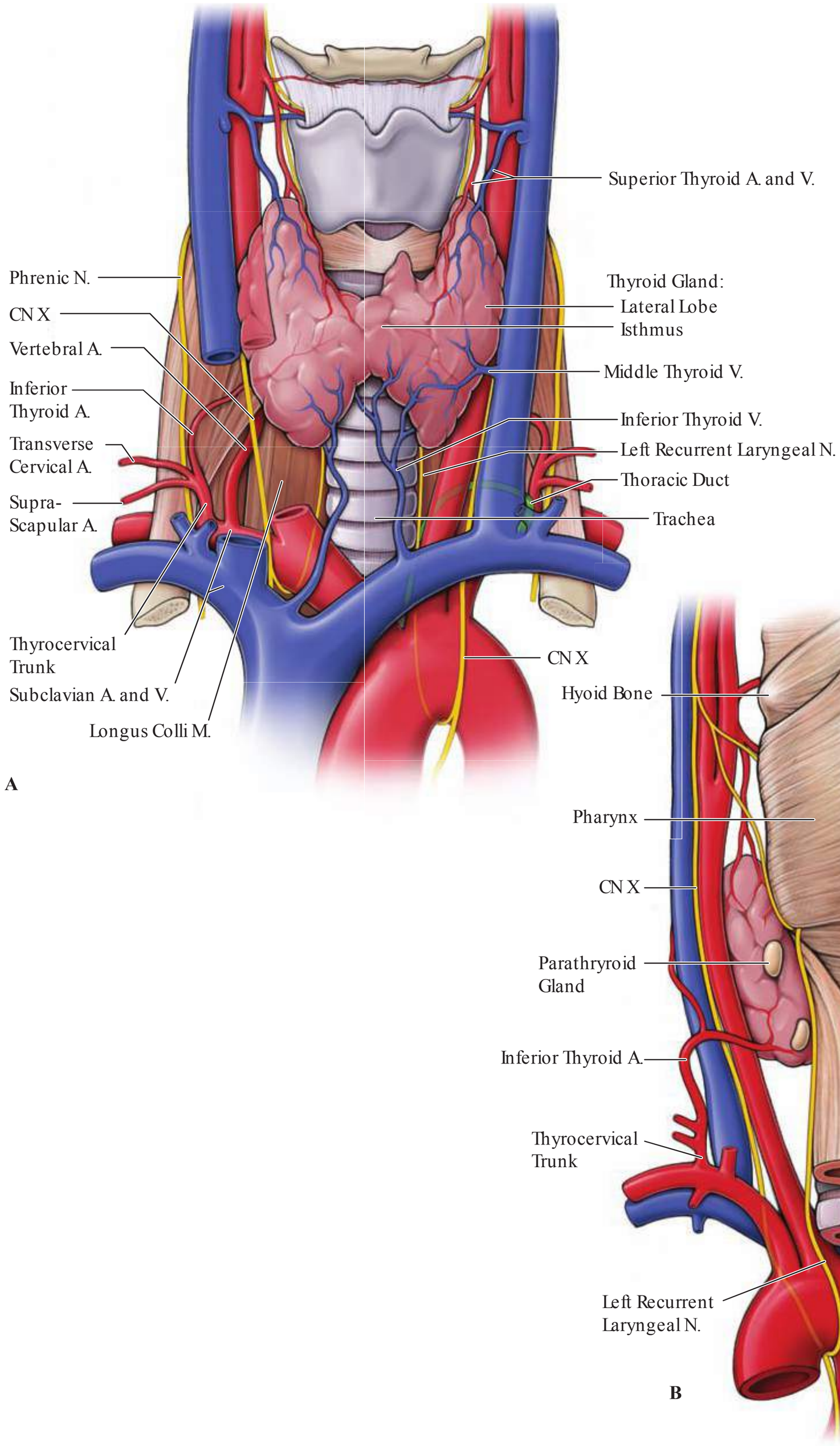


FiGUre 15-1. Thyroid anatomy. (Reproduced, with permission, from Morton DA, Foreman KB, Albertine KH. The Big Picture: Gross Anatomy. New York, NY: McGraw-Hill Education; 2011. Figure 26-3AB.)

vasCulature

See Figure 15-2.

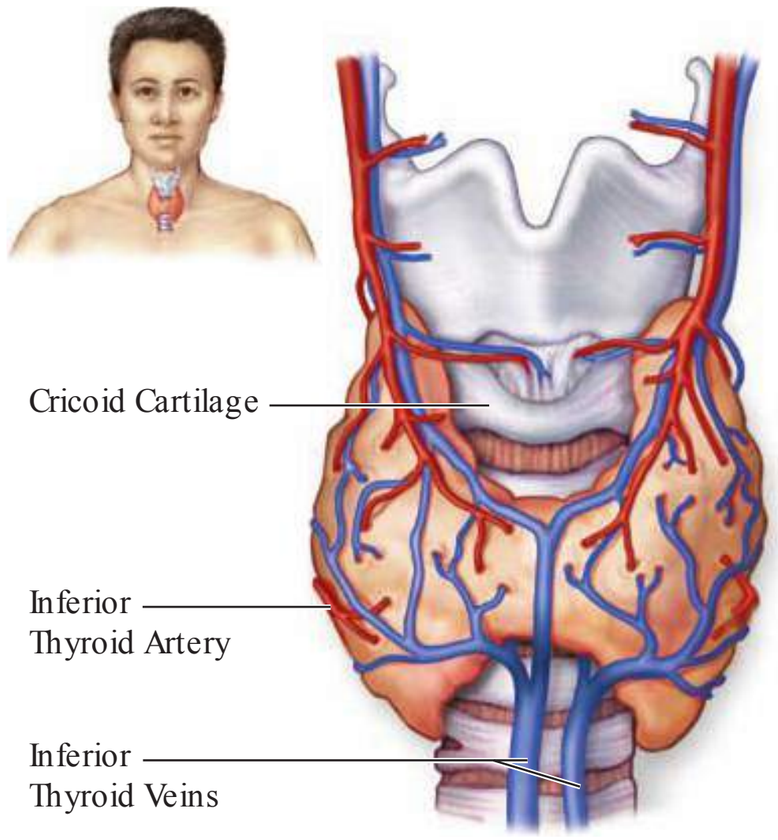


FIGURE 15-2. Thyroid blood supply. (Reproduced, with permission, Mescher AL. Junqueira's Basic Histology. 14th ed. New York, NY: McGraw-Hill Education; 2016. Figure 20-18.)

Arterial

- Superior thyroid arteries (on each side).
  - First branch of external carotid artery at the level of the carotid bifurcation.
- Inferior thyroid artery (on each side).
  - From thyrocervical trunk of subclavian artery.
- Ima (sometimes present).
  - From aortic arch or innominate artery.

Venous

- Superior thyroid vein (on each side).
  - Drains to internal jugular (IJ).
- Middle thyroid vein (on each side).
  - Drains to IJ.
- Inferior thyroid vein (on each side).
  - Drains to brachiocephalic vein.

EXAM TIP

The RLN innervates all the intrinsic muscles of the larynx, except the cricothyroid (supplied by the superior laryngeal nerve), and provides sensory innervation to the mucous membranes below the vocal cord. It can be damaged during a thyroid operation, so the surgeon must know its course well. Damage produces ipsilateral vocal cord paralysis and results in hoarseness or sometimes shortness of breath due to the narrowed airway.

innervation

- The right recurrent laryngeal nerve (RLN) branches from the right vagus nerve, loops under the right subclavian artery, and ascends to the larynx (posterior to the thyroid) between the trachea and esophagus. It may be anterior or posterior to the inferior thyroid artery. The left RLN branches from the left vagus nerve, loops under the aortic arch, and then ascends along the tracheoesophageal groove to the larynx. Both RLNs innervate the muscles of the true vocal cords.
- Sympathetic: Superior and middle cervical sympathetic ganglia (vasomotor).
- Parasympathetic: From vagus nerves, via branches of laryngeal nerves.

hormones

The thyroid gland produces thyroid hormone (TH) using iodide and tyrosine.

hormone regulation

- TSH causes:
  - Increased formation of TH.
  - The release of TH into circulation within 30 minutes.

- The increased TH level in blood then feeds back to the pituitary and results in decreased TSH secretion, by an incompletely understood mechanism.

effects of Thyroid hormone

- Cardiovascular system: Increased heart rate (HR), cardiac output (CO), blood flow, blood volume, pulse pressure (no change in mean arterial pressure [MAP]).
- Respiratory system: Increased respiratory rate (RR), depth of respiration.
- Gastrointestinal (GI) system: Increased motility.
- Central nervous system (CNS): Nervousness, anxiety.
- Musculoskeletal system: Increased reactivity up to a point, then response is weakened; fine motor tremor.
- Sleep: Constant fatigue but decreased ability to sleep.
- Nutrition: Increased basal metabolic rate (BMR), need for vitamins, metabolism of carbohydrate, lipid, and protein; decreased weight.

assessment of function

- If T_4 production is increased, both total T_4 (tT_4) and free T_4 (fT_4) increase.
- If production decreases, both tT_4 and fT_4 decrease.
- If amount of thyroid-binding globulin (TBG) changes, only tT_4 changes, not fT_4.

Congenital anomalies

- Persistent sinus tract remnant of developing gland: **Thyroglossal duct cyst**—may occur anywhere along course as a midline structure with thyroid epithelium, usually between the isthmus and the hyoid bone:
  - Most common congenital anomaly.
  - Few symptoms but may become infected.
  - Easier to see when tongue is sticking out.
  - Surgical treatment: Excision of duct remnant and central portion of hyoid bone (Sistrunk's operation).
- Complete failure to develop.
- Incomplete descent: Lingual or subhyoid position (if gland enlarges, patient will have earlier respiratory symptoms).
- Excessive descent: Substernal thyroid.
- Malformation of branchial pouch.

hyperthyroidism

causes

- Graves' disease.
- Toxic nodular goiter.
- Toxic thyroid adenoma.
- Subacute thyroiditis.
- Functional metastatic thyroid cancer.
- Struma ovarii (abnormal thyroid tissue in ovary).

Graves' disease

- Most common cause of hyperthyroidism in the United States.
- **Mechanism:** Autoimmune disorder that causes an excess of TH to be produced due to the presence of thyroid-stimulating immunoglobulins that stimulates production of TSH.

WARD TIP

Thyroid follicles store enough hormone to last 2–3 months. Thus, there is no need to worry about your postop hypothyroid patients who are NPO for several days. They can resume taking their levothyroxine when they begin a PO diet.

WARD TIP

Palpation of the thyroid is easiest if you stand behind the patient and reach your arms around to the front of the neck. Expect the isthmus to be about one fingerbreadth below the cricoid cartilage. In addition, make sure to palpate for cervical lymph nodes.

WARD TIP

In 70% of cases of lingual thyroid, it is the patient's only functioning thyroid tissue. This means that it is important to look for other functioning thyroid tissue prior to removing a lingual thyroid.

EXAM TIP

Ten percent of patients will have atrial fibrillation that may be refractory to medical treatment until hyperthyroidism is controlled.

- Affects nearly 2% of American women; six times more common in women.
- Onset age 20–40.
- Families with Graves' disease have increased risk of other autoimmune disorders (e.g., diabetes, Addison's) and other thyroid disorders as well.

Signs and Symptoms

- Nervousness, increased sweating, tachycardia, goiter, pretibial myxedema, tremor (90%).
- Heat intolerance, palpitations, fatigue, weight loss, dyspnea, weakness, increased appetite, exophthalmos, thyroid bruit (50–90%).
- Other: Amenorrhea, decreased libido and fertility.

diagnosis

- Labs: Thyroid function tests (TFTs)—increased T_3 and/or T_4 and decreased TSH (negative feedback of ↑ hormone levels).
- Radioactive iodide uptake test (RAIU): Scan shows diffusely increased uptake.

treatment

- Antithyroid drugs.
- Radioiodide ablation with ^{131}I.
- Subtotal or total thyroidectomy.
- Choosing a treatment:
  - Consider: Age, severity, size of gland, surgical risk, treatment side effects, comorbidities.
  - Radioablation is the most common choice in the United States:
    - Indicated for small or medium-sized goiters, if medical therapy has failed, or if other options are contraindicated.
    - Most patients become euthyroid within 2 months.
    - Most patients ultimately require TH replacement (e.g., levothyroxine).
    - Complications include exacerbation of thyroid storm initially.
    - Contraindicated in pregnant patients, women of childbearing age and newborns.
- Surgical treatment:
  - Indicated when radioablation is contraindicated (e.g., young or pregnant patients) or if medical management cannot be used.
  - Patients should be euthyroid prior to excision.
  - Advantage over radioablation is immediate cure.
- Medical therapy:
  - β-blockers provide symptomatic relief.
  - Antithyroid drugs (propylthiouracil [PTU], methimazole) inhibit hormone production and peripheral conversion of $T_4 \rightarrow T_3$.
  - Potassium iodide reduces hormone production (via Wolff–Chaikoff effect), used to shrink gland prior to surgical excision.
  - High recurrence rate with medical therapy.
  - May cause side effects such as rash, fever, or peripheral neuritis.
  - Patients relapse if meds are discontinued.
  - Check TFTs after any treatment to determine if patient is successfully euthyroid or requires hormone replacement.

EXAM TIP

Typical scenario: A 28-year-old female presents with a recent unintentional 10-lb weight loss, and complaining of insomnia despite extreme fatigue. She also complains of amenorrhea and decreased sexual desire. She appears anxious and fidgety during your assessment. On exam, she is tachycardic and with an enlarged thyroid. Laboratory studies reveal TSH of 0.08 and T_4 of 250 nmol/L. Think: Graves' disease. You can control her tachycardia with β-blockers and optimize her for surgery.

Toxic nodular Goiter (Plummer's disease)

- Causes hyperthyroidism but without the extrathyroidal symptoms.
- Treatment is surgical since medical therapy and radioablation has a high failure rate.

- Solitary nodule: Lobectomy.
- Multinodular goiter: Subtotal thyroidectomy.

Thyroid Storm (Thyrotoxicosis)

- Life-threatening extreme exacerbation of hyperthyroidism precipitated by surgery on an inadequately prepared patient (i.e., incomplete β-blockade and noneuthyroid patients), infection, labor, iodide administration, or recent radioablation.
- Patient presents with fever, tachycardia, muscle stiffness or tremor, disorientation/altered mental status.
- Fifty percent of patients with thyroid storm develop congestive heart failure (CHF).
- Has a 20–40% mortality rate.
- Best way to treat this is by avoiding it. Prophylaxis includes achieving euthyroid state preop.
- **Treatment:** Fluids, antithyroid medication (thionamide, PTU, methimazole), β-blockers (propanolol, metoprolol), corticosteroids (hydrocortisone), sodium iodide (NaI) or Lugol's solution (KI), and a cooling blanket.

hypothyroidism

causes

- Autoimmune thyroiditis.
- Iatrogenic: s/p thyroidectomy, s/p radioablation, secondary to antithyroid medications.
- Iodine deficiency.

Signs and Symptoms

Differ, depending on age of diagnosis:

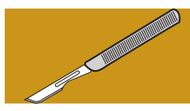
- Infants/peds: Characteristic Down's-like facies, failure to thrive, mental retardation.
- Adolescents/adults (particularly when due to autoimmune thyroiditis):
  - Eighty percent female.
  - Physiologic effects: Bradycardia, decreased CO, hypotension, shortness of breath secondary to effusions.
  - Presentation includes: Fatigue, weight gain, cold intolerance, constipation, menorrhagia, decreased libido and fertility.
  - Less common complaints: Yellow-tinged skin, hair loss, tongue enlargement.

diagnosis

- History and physical exam findings.
- Labs:
  - Decreased T_4, T_3.
  - TSH:
    - Increased in primary hypothyroidism.
  - Decreased in secondary hypothyroidism.
  - Confirm with TRH challenge: TSH will not respond in secondary hypothyroidism.
  - Thyroid autoantibodies present in autoimmune thyroiditis.
  - Low hematocrit (Hct).
- Electrocardiogram (ECG): Decreased voltage and flat or inverted T waves.

Treatment

Thyroxine PO or IV emergently if patient presents in myxedema coma.



OR TIP

Risks of thyroid surgery include recurrent laryngeal nerve injury, hypoparathyroidism, and persistent hyperthyroidism (with subtotal thyroidectomy).



WARD TIP

Immediate treatment (of infants) with thyroid hormone will help minimize the neurologic and intellectual deficits.

Thyroiditis

Acute

- **Infectious etiology:** Streptococcus pyogenes, Staphylococcus aureus, Pneumococcus pneumoniae.
- **Risk factors:** Female sex, goiter, thyroglossal duct.
- **Signs and symptoms:** Unilateral neck pain and fever, euthyroid state, dysphagia.
- **Treatment:** IV antibiotics and surgical drainage.

Subacute (de Quervain's)

- **Etiology:** Post–viral upper respiratory infection (URI).
- **Risk factors:** Female sex.
- **Signs and symptoms:** Fatigue, depression, neck pain, fever, unilateral swelling of thyroid with overlying erythema, firm and tender thyroid, transient hyperthyroidism usually preceding hypothyroid phase.
- **Diagnosis:** Made by history and exam.
- **Treatment:**
  - Usually self-limited disease (within 6 weeks).
  - Manage pain with nonsteroidal anti-inflammatory drugs (NSAIDs).

Chronic (Hashimoto's Thyroiditis)

- **Etiology:** Autoimmune.
- **Risk factors:** Down's syndrome, Turner syndrome, familial Alzheimer's disease, history of radiation therapy as child.
- **Signs and symptoms:** Painless enlargement of thyroid, neck tightness, presence of other autoimmune diseases.
- **Diagnosis:** Made by history, physical, and labs.
  - Labs: Circulating antibodies against microsomal thyroid cell, TH, T_3, T_4, or TSH receptor.
- **Pathology:** Firm, symmetrical, enlargement; follicular and Hürthle cell hyperplasia; lymphocytic and plasma cell infiltrates.
- **Treatment:**
  - Thyroid hormone (usually results in regression of goiter).
  - With failure of medical therapy, partial thyroidectomy is indicated.

Riedel's Fibrosing Thyroiditis

- Rare.
- Fibrosis replaces both lobes and isthmus.
- **Risk factors:** Associated with other fibrosing conditions, like retroperitoneal fibrosis, sclerosing cholangitis.
- **Signs and symptoms:** Usually remain euthyroid; neck pain, possible airway compromise; firm, nontender, enlarged thyroid.
- **Diagnosis:** Open biopsy required to rule out carcinoma or lymphoma.
- **Pathology:** Dense, invasive fibrosis of both lobes and isthmus. May also involve adjacent structures.
- **Treatment:**
  - With airway compromise: Isthmectomy.
  - Without airway compromise: Medical treatment with steroids.

Workup of a Mass

- Fifteen percent of solitary thyroid nodules are malignant.
- If multinodular thyroid gland, risk of malignancy is only 5%.
- Ninety to 95% present as well-differentiated cancer.
- Lateral aberrant thyroid: Usually well-differentiated papillary cancer metastatic to cervical lymph nodes.
- **Risk factors:** History of radiation, family history of thyroid cancer, age, gender.

WARD TIP

The bacteria that cause acute suppurative thyroiditis usually spread through lymphatics from a nearby locus of infection.

WARD TIP

Ten percent of patients with subacute thyroiditis become permanently hypothyroid.

WARD TIP

Twenty percent of patients with Hashimoto's thyroiditis will be hypothyroid at diagnosis. A euthyroid state is more common.

EXAM TIP

Typical scenario: A 35-year-old female who emigrated from Russia in 1990 is referred to you for a solitary right thyroid mass. On exam, you find a solitary mass, 2 × 1 cm, that is firm and fixed. There are no palpable lymph nodes. What is a likely diagnosis? Think: Papillary thyroid cancer, which is the most common variety. Fine-needle aspiration in 85–90% of patients shows cuboidal cells with abundant cytoplasm and psammoma bodies. Computed tomography (CT) shows no other foci of disease. What's your next step? Think: Near-total thyroidectomy.

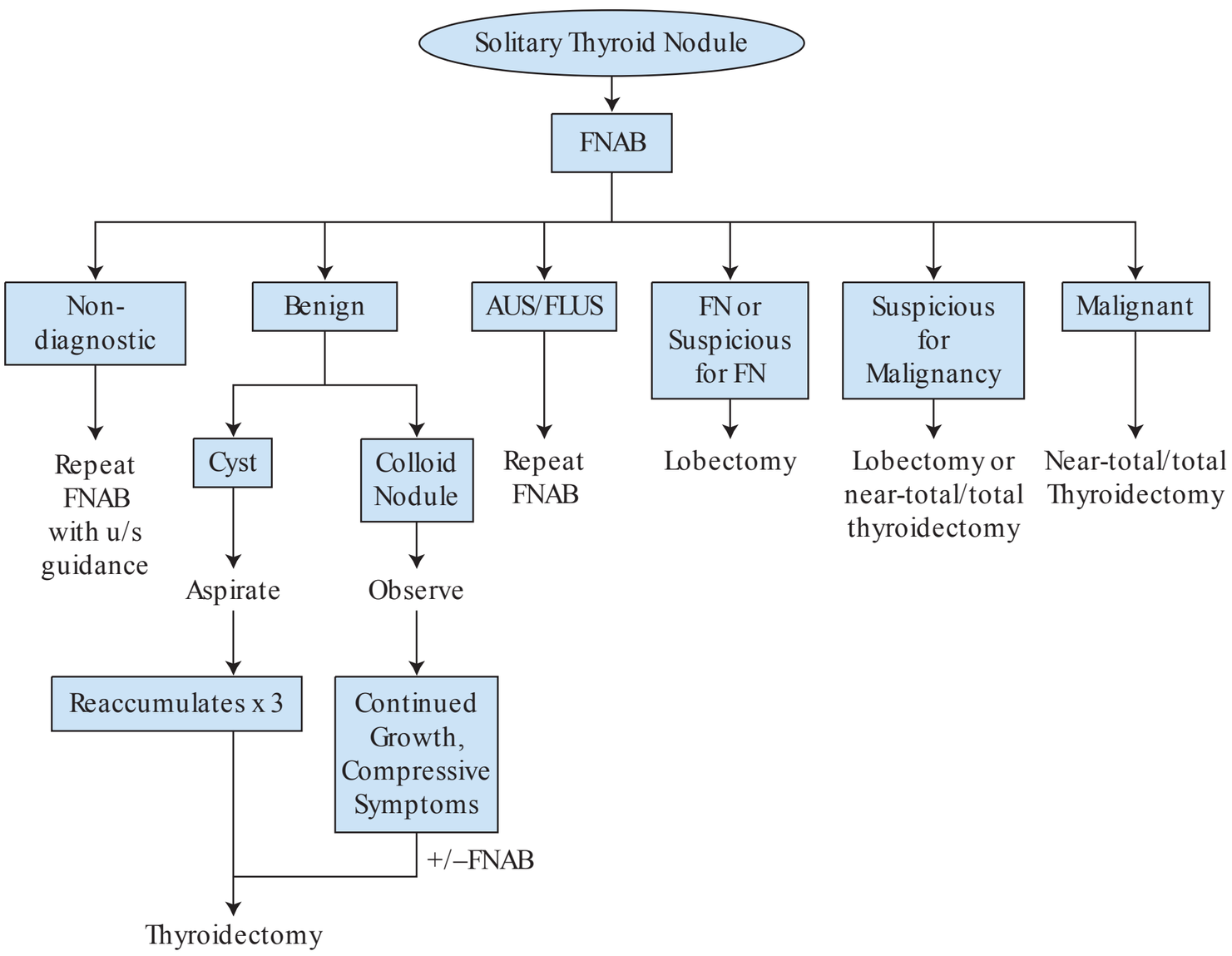


FiGUre 15-3. management of solitary thyroid nodule. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Figure 38-14.)

- **Signs and symptoms:**
  - Voice and/or airway symptoms, sudden enlargement of nodule.
  - History of head and neck radiation therapy leads to 40% risk of developing thyroid cancer, so if patient presents with this history, proceed directly to surgery.
  - Exam: Check size, mobility, quality, adherence of mass, and presence of lymphadenopathy. Concerning findings include hard, fixed gland or palpable cervical lymph nodes.

EXAM TIP

Papillary thyroid cancer is the most Popular (most common type) of thyroid cancers.

diagnosis

Fine-needle aspiration (FNA) is the standard of care for thyroid nodule workup (see Figure 15-3).

- Benign (65%):
  - Ultrasound (US) for sizing and to differentiate nodules and cysts.
  - Obtain thyroglobulin level and follow over time. No need for surgery.
- Suspicious or nondiagnostic (15%): Usually follicular (20% will be malignant).
- Obtain ^{123}I scan:
  - Eighty-five percent are "cold" nodules with a 10–25% chance of malignancy.
  - Five percent are "hot" nodules with only a 1% chance of malignancy.
  - Surgery is indicated if serial T_4 levels fail to regress and future biopsies are worrisome.
- Malignant (15%): Surgery.
- Cyst: Drain completely (curative in 75% of cases). If cyst is > 4 cm, complex, or recurring even after three aspirations, send to OR for removal.

thyroiD CanCer

See Table 15-2.

TABLE 15-2. Thyroid Cancer

	Papillary	Follicular	Medullary	Anaplastic
	Most common type of thyroid cancer	Not common in populations that do not have iodine deficiency		Rarest but worst prognosis
Percent	80–85 (75% of pediatric thyroid cancer)	5–20%	5–10%	1–5%
Risk factors	Radiation	Dyshormonogenesis	MEN II in 30–40%	▪ Prior diagnosis of well-differentiated thyroid cancer ▪ Iodine deficiency
Age group	30–40	40–50	50–60	60–70
Sex (F/M)	2/1	3/1	1.5/1	1.5/1
Signs and symptoms	▪ Painless mass ▪ Dysphagia ▪ Dyspnea ▪ Hoarseness ▪ Euthyroid	▪ Painless mass ▪ Rarely hyperfunctional	▪ Painful mass ▪ Palpable lymph node (LN) (15–20%) ▪ Dysphonia ▪ Dyspnea ▪ Hoarseness	▪ Rapidly enlarging neck mass (large mass at presentation) ▪ Neck pain ▪ Dysphagia ▪ Hard, fixed LN (50%)
Diagnosis	▪ FNA ▪ CT or MRI (to assess local invasion)	▪ FNA ▪ CT or MRI (to assess local invasion)	▪ FNA ▪ Presence of amyloid is diagnostic ▪ Check for calcitonin	▪ FNA
Metastases	Lymphatic (5% at time of presentation)	Hematogenous	▪ Lymphatic (local neck and mediastinal nodes) ▪ Local (into trachea and esophagus)	▪ Aggressive local disease ▪ 30–50% have synchronous pulmonary mets at time of diagnosis
Treatment	▪ Minimal cancer (< 1.5 cm): Limited lobectomy and isthmectomy ▪ Other: Total or near-total thyroidectomy for cancers > 1.5 cm ▪ For + LN, modified radical neck dissection ▪ ^{131}I ablation or thyroid suppression (with thyroid hormone) for patients with residual thyroid tissue or LN mets	▪ Minimal cancer (< 4 cm): Lobectomy and isthmectomy ▪ Other: Total or near-total thyroidectomy for cancers > 4 cm ▪ For + LN, modified radical neck dissection ▪ ^{131}I ablation for patients with residual thyroid tissue or LN mets	▪ Sporadic (80%): Total thyroidectomy ▪ Familial (20%): Total thyroidectomy and central neck node dissection ▪ No value for ^{131}I ablation ▪ Follow patients with calcitonin levels	▪ Debulking resection of thyroid gland and adjacent structures ▪ External radiation therapy (XRT) ▪ Doxorubicin-based chemotherapy

(continues)

TABLE 15-2. Thyroid cancer (continued)

	Papillary	Follicular	Medullary	Anaplastic
Prognosis	▪ Worse for older patients and those with distant mets ▪ Presence of + LN not strongly correlated with overall survival	▪ Worse for older patients, those with distant mets, tumor size > 4 cm, high tumor grade ▪ Presence of + LN not strongly correlated with overall survival		Poor prognosis
Survival	10-year survival: 74–93%	10-year survival: 60–80%	10-year survival: 70–80%	Median survival: 4–5 months

Parathyroid

Development

- Superior parathyroid glands (2) develop from fourth pharyngeal pouch.
- Inferior parathyroid glands (2) develop from third pharyngeal pouch.

anatomy

- Weight < 50 mg/gland.
- 3 × 3 × 3 mm.
- Adult position of superior gland constant and next to the superior lobes of thyroid.
- Inferior glands have more variable position (posterior/lateral to thyroid and below inferior thyroid artery).
- Not uncommon for one of the inferior glands to be "missing."
- Most common location in thymus. Other sites include intravagal, groove in carotid sheath.
- Yellow-brown tissue similar to surrounding fatty tissue.
- Histology: Normal gland contains mainly chief cells (produce parathyroid hormone [PTH]), with occasional oxyphils.
- Vasculature: Inferior thyroid arteries; superior, middle, and inferior thyroid veins.

physiology/Calciumhomeostasis

- **Primary function:** Regulation of calcium and phosphate metabolism by PTH, vitamin D, and calcitonin.
- Primary organ systems involved: GI tract, bone, kidney.

pth

- Synthesized by the parathyroid glands (see Figure 15-4).
- Serum calcium levels regulate secretion of cleaved PTH by negative feedback mechanism.
- Bone:
  - Stimulates osteoclasts (increased bone resorption).

WARD TIP

FNA is less reliable if patient has history of irradiation, and initial OR biopsy may be appropriate.

EXAM TIP

Of the half of serum calcium in an ionized, active form, 80% is bound to albumin, and 20% is found in a citrate complex.

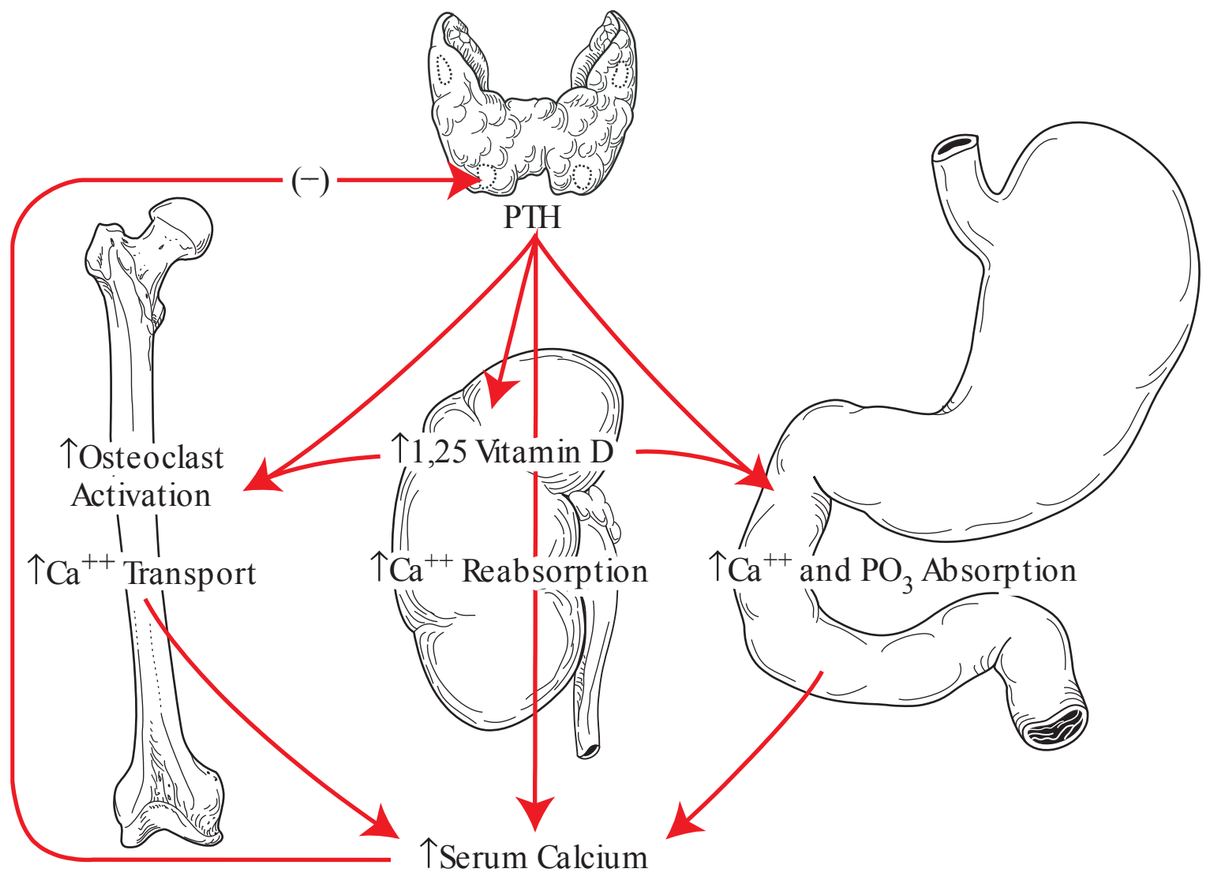


FIGURE 15-4. Actions of parathyroid hormone.

WARD TIP

Hypercalcemic crisis (calcium > 13 mg/dL and symptomatic): Treat with saline, furosemide, bisphosphonates, and, if needed, antiarrhythmic agents.

EXAM TIP

Calcitonin tones down Ca^{2+} levels.

EXAM TIP

Hyperparathyroidism = ↑ Ca^{2+}. Think: Stones, bones, groans, and moans.

- Inhibits osteoblasts (stops bone production).
- This causes release of calcium and phosphate.

- Kidney:
  - Increases reabsorption of calcium.
  - Increases phosphate excretion.
- GI tract:
  - Stimulates hydroxylation of 25-OH D → 1,25 OH D.
  - 1,25 OH D (vitamin D) increases the intestinal absorption of dietary calcium (primarily in duodenum) and phosphate.

Calcitonin

- Secreted by thyroid C cells.
- Inhibits bone resorption (inhibits calcium release).
- Increases urinary excretion of calcium and phosphate.
- Works as a counterregulatory hormone to PTH.

hyperparathyroidism

Primary

Due to overproduction of PTH, causing increased absorption of calcium from intestines, increased vitamin D_3 production, and decreased renal calcium excretion, thereby raising the overall serum level of calcium and lowering the amount of phosphorus.

- **Incidence:** 1/4,000 in the United States.
- **Risk factors:** MEN I, MEN IIA, history of radiation.
- **Signs and symptoms:**
  - "Stones": Kidney stones.
  - "Bones": Bone pain, pathologic fractures, subperiosteal resorption.
  - "Groans": Nausea, vomiting, muscle pain, constipation, pancreatitis, peptic ulcer disease.
  - "Moans": Lethargy, confusion, depression, paranoia.
- **Etiology:**
  - Solitary adenoma: 85–90%
  - Four-gland hyperplasia: 10%
  - Cancer: < 1%

- Preop imaging: Ultrasound, Sestamibi scan, CT/magnetic resonance imaging (MRI), operative exploration.
- **Diagnosis:** Elevation of plasma PTH, with inappropriately high serum calcium. Check urine for calcium to rule out diagnosis of familial hypocalciuric hypercalcemia (will be low if familial disease, and high if primary hyperparathyroidism).
- **Treatment:**
  - Solitary adenoma: Solitary parathyroidectomy with neck exploration to identify and possibly biopsy 3 remaining glands. If preop sestamibi scan is done to localize area, no need for neck exploration.
  - Multiple gland hyperplasia: Remove three glands, or all four with reimplantation of at least 30 g of parathyroid tissue in forearm or other accessible site to retain function (this makes it easier to resect additional parathyroid gland if hyperparathyroid state persists).
  - Outcome:
    - First operation has 98% success rate.
    - Reoperation has 90% success rate if remaining gland is localized preop.

Secondary

Increased PTH due to hypocalcemia that is the result of chronic renal failure (phosphate retention → low calcium), GI malabsorption, osteomalacia, or rickets.

- **Signs and symptoms:**
  - Bone pain from renal osteodystrophy and pruritus.
  - Patients are often asymptomatic.
- **Diagnosis:** Made by labs in asymptomatic patient; usually due to four-gland hyperplasia.
- **Treatment:**
  - Nonsurgical: In renal failure patients, correct calcium and phosphate.
    - Restrict phosphorus intake, treat with phosphorus-binding agents and calcium/vitamin D supplementation. Adjust dialysate to maximize calcium and minimize aluminum.
  - Surgical: Indicated for intractable bone pain or pruritus, or pathologic fractures, with failure of medical therapy. No role for parathyroid surgery in secondary HPTH.
    - Perform renal transplant if necessary.

Tertiary

Due to persistent hyperparathyroidism after treatment for secondary hyperparathyroidism. Due to autonomously functioning parathyroid glands that are resistant to negative feedback from high calcium levels. Usually s/p renal transplant.

- Usually a short-lived phenomenon.
- If persistent, surgery is indicated (3½-gland parathyroidectomy).

hypoparathyroidism

- Uncommon.

etiology

- Surgically induced: Following total thyroidectomy; usually transient and treated if symptoms develop.
- Congenital absence of all four glands.
- DiGeorge syndrome: Absence of parathyroid and thymus.
- Functional: Chronic hypomagnesemia.

EXAM TIP

osteitis fibrosa cystica

- Caused by hyperparathyroidism, characterized by painful, cyst-like brown tumor infiltrating the bone. Tumors regress with management of hyperparathyroidism.

WARD TIP

Not all patients with hypercalcemia have hyperparathyroidism. Hypercalcemia of malignancy (due to tumor-secreted PTH-related protein) must be ruled out. Malignancies commonly implicated include colon, lung, breast, prostate, head, neck, and multiple myeloma.

EXAM TIP

Patients with familial hyperparathyroidism (i.e., MEN I/IIa) have a high recurrence rate; total parathyroidectomy with forearm reimplantation is indicated to facilitate potential reoperation if HPTH persists. Patients with sporadic four-gland hyperplasia may undergo total parathyroidectomy with reimplantation or three-gland excision.

EXAM TIP

Typical scenario: A patient complains of tingling around her lips on postoperative day 1 s/p total thyroidectomy. Think: Iatrogenic hypoparathyroidism causing hypocalcemia. Supplement calcium and vitamin D. Continue to follow calcium levels.

Signs and Symptoms

- Numbness and tingling of circumoral area, fingers, toes.
- Anxiety, confusion.
- May progress to tetany, hyperventilation, seizures, heart block.

Treatment

- Supplementation with PO calcium and vitamin D (to help GI absorption).
- Pseudohypoparathyroidism: Familial disease causing resistance of PTH at target tissue. Patients remain hypocalcemic and hyperphosphatemic despite bone resorption from elevated PTH. Treatment consists of calcium and vitamin D supplementation.

parathyroid cancer

EXAM TIP

hypocalcemia
Chvostek: Contraction of facial muscles when tapping on facial nerve.
Trousseau: Development of carpal spasm by occluding blood flow to forearm.

Signs and Symptoms

- Forty to 50% present with firm, fixed mass that is palpable.
- Extremely high calcium and PTH levels. Usually has high levels of human chorionic gonadotropin (hCG—tumor marker).
- Neck pain, voice change (due to lesion in RLN).

Treatment

- En bloc surgical resection of mass and surrounding structures, along with ipsilateral thyroid lobectomy, and ipsilateral lymph node dissection.
- Postop external radiation therapy (XRT) and chemotherapy are not usually beneficial.
- Postop complications: RLN damage, severe hypocalcemia (hungry bone syndrome).
- Five-year survival: 70%.

Adrenal Gland

anatomy

- Bilateral retroperitoneal organs, anterior and medial to superior pole of kidneys.
- At level of T11.
- Size: 3–6 g, 5 × 2.5 cm.
- Vasculature: Branches of aorta, inferior phrenic, and renal arteries.
- Venous drainage: Right side drains to inferior vena cava (IVC), and left side drains to the left renal vein.

histology

- Cortex.
- Glomerulosa: 15%; aldosterone synthesis.
- Fasciculata: 75%; steroids and cortisol synthesis.
- Reticularis: 10%; cortisol, androgen, and estrogen secretion.

physiology

See section on endocrine organs and Figure 15-5.

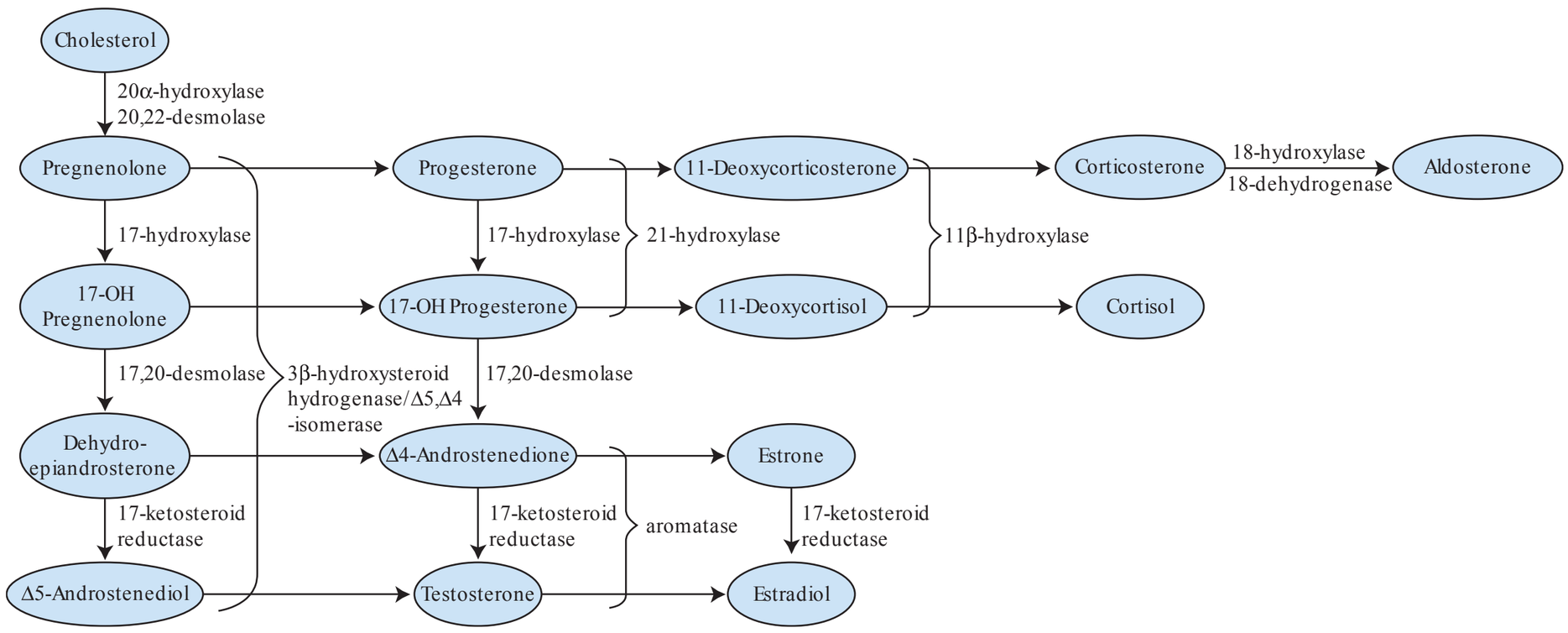


FIGURE 15-5. Pathways of adrenal steroid synthesis.

hyperplasia

Area of cortex expands and becomes hyperfunctioning.

adenoma

- Most are unilateral.
- If adrenal gland < 6 cm, usually observe unless hormonally active (↑ cortisol, ↑ ACTH) or increasing in size.
- If adrenal gland > 6 cm, surgically resect due to increased risk of adrenocortical carcinoma.

adrenal cortical carcinoma

definition

Very rare adrenal tumor.

epidemiology

Affects women more than men, peak < 5 years old or 30–40 years old.

Signs and Symptoms

- Vague abdominal complaints (due to enlarging retroperitoneal mass).
- Symptoms related to overproduction of a steroid hormone (most tumors are functional).

diagnosis

- Twenty-four-hour urine collection for cortisol, aldosterone, catecholamines, metanephrine, vanillylmandelic acid (VMA), 17-OH corticosteroids, 17-ketosteroids.
- CT (lesions > 7 mm) or MRI (especially for assessing IVC invasion).
- Chest x-ray (CXR) to rule out pulmonary metastases.

Treatment

- Radical en bloc resection, but only one third of adrenal carcinomas are operable.
- If resection cannot be completed, debulk to reduce amount of cortisol-secreting tissue.

EXAM TIP

Fifty percent of adrenal cortical carcinomas secrete cortisol, resulting in Cushing's syndrome.

- Bone metastases should be palliated with XRT.
- No role for chemotherapy.
- Monitor steroid hormone levels postop.
- Recurrence also warrants resection.
- Recurrence: Lungs, lymph nodes, liver, peritoneum, bone.
  - In 10% of bilateral adrenalectomies, patients may develop Nelson's syndrome—excess production of ACTH from pituitary adenoma. Pituitary adenoma often causes visual disturbances (due to mass effect on chiasm), hyperpigmentation (↑ melanocyte-stimulating hormone [MSH]), and amenorrhea as well.

Prognosis

- Seventy percent present in stage III or IV.
- Five-year survival 40% for complete resection.
- If local invasion, median survival time is 2–3 years.

Cushing's syndrome

definition

Excessive cortisol production.

causes

- Iatrogenic administration of corticosteroids most common cause!
- Pituitary tumor that secretes ACTH (i.e., Cushing's disease).
- Ectopic ACTH secretion by tumor elsewhere stimulates adrenal cortisol production.
- Adrenal tumor that secretes cortisol.

Signs and Symptoms

Appearance

- Weight gain
- Truncal obesity
- Extremity wasting
- Buffalo hump
- Moon facies
- Acne
- Purple striae
- Hirsutism

Physiologic

- Mild glucose intolerance
- Amenorrhea
- Decreased libido
- Depression
- Impaired memory
- Muscle weakness

EXAM TIP

Typical scenario: A young male presents with weight gain, especially in the trunk; loss of muscle mass; and a buffalo hump. He has recently been noted to be mildly glucose intolerant. His past medical history is significant for severe asthma, for which he is chronically on steroids. Think: Cushing's syndrome secondary to exogenous administration of steroids.

diagnosis

See Table 15-3.

- Confirm presence of hypercortisolism:
  - Low-dose dexamethasone test.
  - Twenty-four-hour urinary cortisol looking for cortisol or its metabolites (e.g., 17-hydroxycorticosteriods).
  - Direct measurement of serum cortisol.

TABLE 15-3. Assays in the Workup of Cushing's Syndrome

Test	Result	Interpretation
24-hour urinary free cortisol or single dose dexamethasone suppression test	Normal	Hypercortisolism can be ruled out: ▪ Low-dose dexamethasone (2 mg) normally decreases urinary cortisol levels in normal patients. Lack of suppression confirms hypercortisolism ▪ High-dose dexamethasone (8 mg) will only decrease urinary cortisol if pituitary-dependent cause of ↑ ACTH (Cushing's disease), but will not suppress cortisol at all if cause is either ectopic ACTH production or a primary adrenal tumor
Plasma ACTH	Very high Intermediate value Low or undetectable	Ectopic ACTH production Pituitary tumor Adrenal source: Either adenoma, hyperplasia, or, very rarely, cancer
Urinary 17-ketosteroids	< 10 mg/day > 60 mg/day In between	Adenoma Cancer Likely hyperplasia
Metyrapone test	Increased ACTH	Pituitary cause
Petrosal sinus sampling	Sinus/plasma ACTH ratio > 3 after corticotropin-releasing hormone (CRH) administration	Identifies Cushing's disease with 100% sensitivity

- Determine whether cortisol production is pituitary dependent or independent: High-dose dexamethasone test.
- CT (adrenals): Can distinguish cortical hyperplasia from tumor with sensitivity > 95%, but lacks high specificity.
- MRI (sella).
- Petrosal sinus sampling (will see elevated ACTH if pituitary tumor).
- Adrenal cortisol production: High cortisol, low plasma ACTH (due to negative feedback from cortisol production), and no suppression of cortisol with high-dose dexamethasone suppression test.
- Ectopic ACTH production: Increased plasma cortisol and ACTH, and no suppression of cortisol (because only ACTH from pituitary, and not ectopic source, is capable of negative feedback).
- Pituitary tumor (Cushing's disease): Mild elevation of ACTH and cortisol is successfully suppressed with high-dose dexamethasone (negative feedback on pituitary).

Treatment

- Cushing's disease: Transsphenoidal resection of pituitary adenoma.
- Adrenal adenoma: Adrenalectomy.
- Adrenal carcinoma: Adrenalectomy.
- Ectopic ACTH: Resection of primary lesion (usually in lung).
- Unresectable lesions and recurrence should be debulked for palliation.
- Medical treatment to suppress cortisol production: Metyrapone (inhibits cortisol production), aminoglutethimide, mitotane, ketoconazole.

EXAM TIP

Most common cause of ectopic ACTH production is small cell lung cancer (a.k.a. oat cell carcinoma), followed by carcinoid tumors.

WARD TIP

Low-dose dexamethasone suppression test: Single dose of steroid at 11 p.m., followed by measurement of serum and urinary cortisol levels at 8 a.m.

Normal: < 5 µg/dL (because evening dose suppresses further release).

Abnormal: > 5 µg/dL high-dose dexamethasone (8 mg) distinguishes pituitary cause (suppression) from adrenal or ectopic cause (no suppression).

WARD TIP

Most common cause of secondary adrenal insuf ciency is iatrogenic due to long-term glucocorticoid therapy.

aDDison's Disease

definition

Adrenal insuff ciency:

- Primary: Due to destruction of adrenal cortex with sparing of medulla.
- Secondary: Failure due to hypothalamic or pituitary abnormalities.

etiology

Primary

- Post-adrenalectomy.
- Autoimmune adrenalitis.
- Tuberculosis (TB).
- Fungal infection.
- Acquired immune deficiency syndrome (AIDS).
- Metastatic cancer.
- Familial glucocorticoid deficiency.

Secondary

- Exogenous steroids.
- Craniopharyngioma.
- Pituitary surgery or irradiation.
- Empty sella syndrome.

Addisonian Crisis

- Acute situation due to some extrinsic stressor (i.e., infection, surgery).

EXAM TIP

The sudden cessation of long-term glucocorticoid therapy can precipitate adrenal insuf ciency because it suppresses the intrinsic control by the hypothalamus and pituitary. Six months may be required for the intrinsic controls to return to normal.

EXAM TIP

Typical scenario: A patient with known Addison's disease presents with acute upper abdominal pain, with peritoneal signs and confusion. Think: Addisonian crisis.

Signs and Symptoms

- Nausea.
- Vomiting.
- Abdominal pain.
- Tachycardia.
- Weight loss.
- Weakness.
- Fatigue.
- Lethargy.
- Hyperpigmentation (low levels of cortisol cause ↑ pituitary production of proopiomelanocortin [POMC], which is precursor to both ACTH as well as MSH).
- Fever and hypovolemic shock in Addisonian crisis.

diagnosis

- Hyponatremia, hyperkalemia (due to ↓ adosterone, which is normally produced in adrenals).
- ACTH stimulation test: Give ACTH and measure cortisol level after 30 minutes. If adrenal failure is present, there will be no increase in cortisol.
- Baseline ACTH level is elevated in patients with primary failure due to absence of negative feedback.

OR TIP

Patients with Addison's disease or who have been taking exogenous steroids for 6 months or longer are likely to require stress-dose steroids perioperatively. The timing and dose may vary depending on the planned procedure and baseline doses of the patient.

Treatment

- Glucocorticoid therapy for primary and secondary causes.
- Additional mineralocorticoid therapy for primary cause.
- Addisonian crisis: Volume (D_5NS) and glucocorticoids IV.

hyperaldosteronism

definition

- Hyperaldosteronism
- Primary
- Secondary

etiology

- Primary (due to excessive aldosterone secretion): Conn's syndrome:
  - Aldosterone-secreting tumor (66%)
  - Idiopathic adrenocortical hyperplasia (30%)
- Secondary (due to elevated renin → elevated aldosterone):
  - Renal artery stenosis
  - Cirrhosis
  - CHF
  - Normal pregnancy

Signs and Symptoms

- Hypertension
- Muscle weakness and cramping
- Headache
- Polyuria
- Polydipsia
- Hypokalemia

diagnosis

- **Primary/Conn's syndrome:**
  - Diastolic hypertension without edema.
  - Elevated plasma aldosterone.
  - Normals or low plasma renin.
  - Hypokalemia, elevated urinary potassium (off antihypertensive medications).
  - Post-captopril plasma aldosterone:
    - Normally results in decreased aldosterone.
    - Diagnostic of hyperaldosteronism if ratio > 50.
  - Imaging:
    - CT picks up tumors > 1 cm. If there is an aldosteronoma, opposite adrenal appears atrophied.
    - Iodocholesterol scan: Picks up 90% of aldosteronomas and shows how functional they are. Hyperplasia will present as bilateral hyperfunction versus unilateral (for tumor).
    - If all imaging is nondiagnostic, then sample adrenal vein for aldosterone and cortisol pre- and post-ACTH.
    - Unilateral elevation of aldosterone or aldosterone/cortisol ratio indicates aldosterone-secreting adenoma.
    - Bilateral elevation of aldosterone is consistent with hyperplasia.

Treatment

- Hyperplasia: Medical treatment with spironolactone, nifedipine, amiloride, and/or other antihypertensive. **No surgery!**
  - Adenoma: Laparoscopic adrenalectomy.
  - Outcome:
    - Most patients become normotensive and normokalemic with treatment.
    - Twenty to 30% have recurrent hypertension (HTN) in 2–3 years, but for unknown reason.
- For secondary hyperaldosterone, treat underlying cause.

WARD TIP

Renin is produced in the juxtaglomerular (JG) cells of the kidney when blood pressure is low, and stimulates conversion of angiotensinogen to angiotensin I in the kidney. Angiotensin I is converted to angiotensin II in the lung. Angiotensin II causes adrenals to produce aldosterone.

WARD TIP

In working up a patient for suspected Conn's syndrome, make sure it isn't just a patient with uncontrolled hypertension on potassium-wasting diuretics.

hypoaldosteronism

definition

Decreased aldosterone without a change in cortisol production.

etiology

- Congenital error of aldosterone synthesis.
- Failure of zona glomerulosa (autoimmune).
- Status post adrenalectomy.
- Drug inhibition.

Signs and Symptoms

- Postural hypotension
- Persistent severe hyperkalemia
- Muscle weakness
- Arrhythmia

Treatment

Mineralocorticoid therapy.

Medullary Tumors

neuroblastoma

definition

- Embryonal neural crest tumor occurring primarily in children (small round blue cell tumor).
- Fourth most common pediatric malignancy.
- Can occur anywhere along sympathetic chain—50% in adrenal, 25% in paraspinal ganglia, 20% in thorax, 5% in pelvis.
- May spontaneously differentiate and regress.
- Aggressive tumor that commonly presents with distant metastases, in 50% of infants and 66% of older children (to lymph node, bone, liver, subcutaneous tissue).

Associated diseases

- Neurofibromatosis
- Beckwith–Wiedemann syndrome
- Trisomy 18

Signs and Symptoms

- Abdominal or flank mass.
- Respiratory distress.
- Subcutaneous blue tumor nodules (blueberry muffin sign).

diagnosis

- Imaging: CT for staging, MRI.
- Urinary tumor markers: Elevated 24-hour levels of homovanillic acid (HVA), VMA, and metanephrines.

EXAM TIP

Typical scenario: A 50-year-old male has an abdominal CT scan. Appendicitis is confirmed, and the patient undergoes a successful appendectomy and uneventful recovery. The CT scan also revealed a 2-cm adrenal mass. What's your next step? Think: Test for functionality. If the mass is functional, it should be resected regardless of its small size.

Treatment

- Localized disease (stages I and II): Surgical resection.
- Nonlocalized disease (stage II): Surgical resection with chemotherapy + /– radiation.
- Metastatic disease (stage IV): Surgical resection with chemotherapy + /– radiation.
- Lymph node metastasis warrants XRT.

Prognosis

- Mortality lower when diagnosed within first year of life.
- Five-year survival 90% for disease confined to primary site; 20–40% for disseminated disease.

pheochromocytoma

definition

Chromaffin cell tumor that is most commonly in the adrenal medulla (> 90%), but may be anywhere along the sympathetic chain. Most common site for extra-adrenal pheochromocytoma is the organ of Zuckerkandl.

Associated risk Factors

- MEN IIA and IIB (usually result in bilateral adrenal tumors)
- Von Hippel–Lindau disease
- Neurofibromatosis
- Family history

Signs and Symptoms

- HTN: May be sustained elevation, normal with paroxysmal HTN, or sustained HTN with acute elevations. Most common presenting symptom.
- Headaches.
- Anxiety, palpitations, pallor, diaphoresis.

diagnosis

- Twenty-four-hour urine collection for catecholamine by-products: VMA, metanephrine, normetanephrine.
- Serum epinephrine and norepinephrine (note: if elevated epinephrine, must be adrenal tumor).
- Clonidine test: Will suppress plasma catecholamine concentrations in normal patients but not in patients with pheochromocytoma.
- CT or MRI, positron emission tomography (PET), or nuclear scan (^{131}I-metaiodobenzylguanidine).

Treatment

- Preop α-adrenergic blockade (phenoxybenzamine) followed by β-blockers for persistent tachycardia.
- Intraoperative arterial blood pressure monitoring is essential because extreme changes in blood pressure may occur with manipulation.
- Important to ligate veins first to prevent unintentional release of catecholamines that may result from manipulation of adrenal gland.
- If malignant:
  - Resect recurrences and metastases when they occur.
  - Treat with catecholamine blockade.
  - Use XRT for bony mets.
  - Chemotherapy has a 60% response rate.

WARD TIP

Most common extra-adrenal location is organ of Zuckerkandl (to left of aortic bifurcation at inferior mesenteric artery).

EXAM TIP

Pheochromocytoma
- If extra-adrenal, then more likely malignant
- If bilateral, more likely familial

WARD TIP

- 10% rule for pheochromocytoma:
  - 10% malignant
  - 10% familial
  - 10% extra-adrenal
  - 10% bilateral
  - 10% in children

WARD TIP

Alpha blockade must precede beta blockade in patients with pheochromocytoma. The use of β-blockers will cause negative inotropic effects and result in unopposed α-induced vasoconstriction, which may precipitate malignant hypertension and cardiac failure.

Prognosis

For malignant tumors: 5-year survival 36–60%.

Pituitary

anatoMy

See Figure 15-6.

- Ectodermal origin.
- Relations:
  - Within sella turcica (floor of sella is roof of sphenoid sinus).
  - Surrounded by dura.
  - Optic chiasm superior and anterior to pituitary stalk.
  - Lateral cavernous sinuses.
  - Superior: Diaphragma sella (meningeal tissue).
  - Size: 12 × 9 × 6 mm, 500–600 mg.
- Parts:
  - Adenohypophysis: Anterior lobe.
  - Neurohypophysis: Posterior lobe.
- Blood supply:
  - Anterior lobe lacks a direct supply. Portal channels from the hypothalamus and posterior pituitary supply it.
  - Posterior pituitary is supplied by middle and inferior hypophyseal arteries, branches of the internal carotid artery.
  - Drains via cavernous sinuses to petrosal sinuses to jugular veins.
- For hormone actions, see section on endocrine organs.

aDenoMa

- Benign tumor arising from anterior lobe.
- Divided into two types based on size:
  - Macroadenoma: > 1 cm diameter.
  - Microadenoma: < 1 cm diameter.

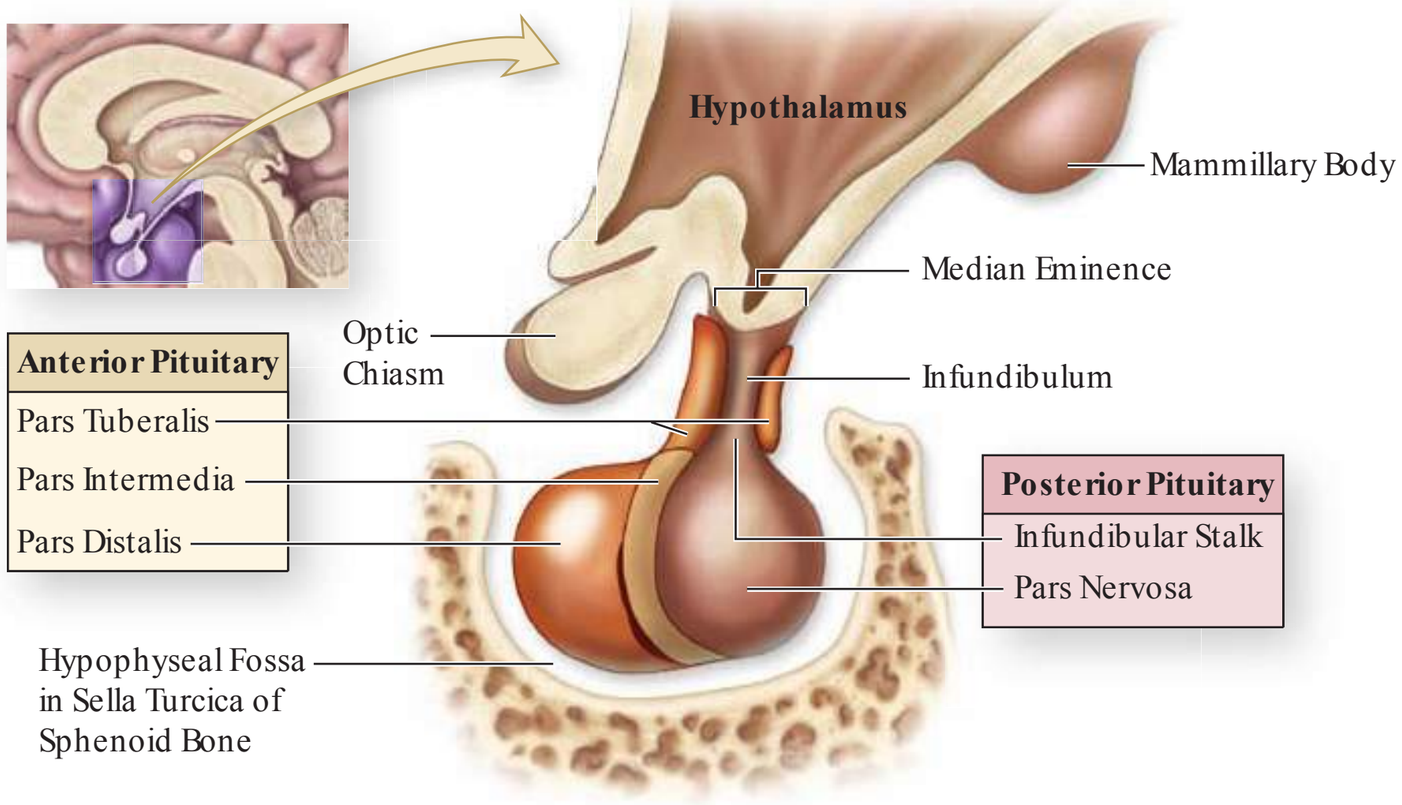


FiGUre 15-6. Pituitary anatomy. (Reproduced, with permission, Mescher AL. Junqueira's Basic Histology. 14th ed. New York, NY: McGraw-Hill Education; 2016. Figure 20-2.)

Signs and Symptoms

- Macroadenomas: Visual loss (bilateral hemianopsia), hypopituitarism, headache, hyperprolactinemia due to compression of surrounding structures.
- Microadenomas: Signs and symptoms depend on type of hormone overproduced.
- Prolactinoma (most common): Secondary amenorrhea, galactorrhea.
- Growth hormone-producing tumor: Gigantism or acromegaly depending on age of patient; also, coarse facial features, thick finger and heel pads, cardiomegaly, hepatomegaly, enlarged mandible, increased teeth spaces, neuropathy, arthropathy, osteoporosis, HTN, diabetes mellitus (DM), goiter.
- ACTH: See Adrenal section.
- Multihormonal.

diagnosis

Imaging and clinical presentation.

Treatment

- Preoperative: Complete endocrine assessment; electrolytes to look for borderline diabetes insipidus.
- Surgery:
  - Transsphenoidal approach: Results in improved function of remaining pituitary gland.
  - Transcranial approach: When transsphenoidal not possible due to location of carotid arteries, extrasellar tumor.
  - Perioperative glucocorticoids, serial visual field assessment, repeat endocrine assessment.
  - Postop XRT: For large lesions.
- Primary radiation therapy: Consider when surgery contraindicated for other reasons in nonfunctioning tumor as primary therapy may worsen preexisting hypopituitarism.
- Medical treatment:
  - For prolactinomas: Bromocriptine.
  - For GH-secreting adenomas: Somatostatin, which decreases tumor size in 20–50%, normalizes GH in 50%, and normalizes IGF-1 in 40–80%.

complications

- Death due to direct hypothalamic injury (< 1%).
- Delayed mortality: Cerebrospinal fluid (CSF) leak, vascular injury.
- Morbidity: Diabetes insipidus (2–17%), cerebrovascular accident (CVA), meningitis.

sheehan's syndrome

Postpartum infarction and necrosis of pituitary leading to hormonal failure.

etiology

Pituitary ischemia due to hemorrhage, hypovolemic shock, pituitary portal venous thrombosis.

Signs and Symptoms

- Failure of lactation.
- Amenorrhea.
- Progressive decreased adrenal function and thyroid function.



WARD TIP

Macroadenomas compress the pituitary stalk, preventing dopamine from traveling from hypothalamus to posterior pituitary gland where it normally inhibits prolactin production. Thus, compression results in hyperprolactinemia (i.e., nipple discharge).

posterior pituitary Disorders

Syndrome of inappropriate Antidiuretic hormone (SiAdh)

- Occurs in 15% of hospital patients.
- Impaired water secretion.
- Hypersecretion of ADH results in increased urinary sodium with elevation of urine osmolality.
- Causes: CNS injury, cancer, trauma, drugs.

diabetes insipidus

- Decreased ADH secretion.
- Impaired water conservation; large volumes of urine, leads to increased plasma osmolality and thirst.
- One third idiopathic; two thirds due to tumor or trauma.

The Spleen

Description 258

Anatomic Boundaries 258

Splenectomy 258

- Types 258
- Complications 258

Tumors of the Spleen 258

Splenic Injury 261

- Signs and Symptoms 261
- Treatment 261

Splenic Abscess 262

- Causes 262
- Signs and Symptoms 263
- diagnosis 263
- Treatment 263
- Complications 263

WARD TIP

Twenty percent of patients have an accessory spleen.

WARD TIP

Most common indications for splenectomy:

1. Trauma
2. Idiopathic thrombocytopenic purpura (ITP) refractory to steroids

EXAM TIP

Conditions associated with rupture of the spleen:

- Blunt LUQ trauma
- Mononucleosis
- Malaria
- Splenic abscess

WARD TIP

Escherichia coli O157:H7 is an invasive gastroenteritis resulting in hemolytic-uremic syndrome (HUS). HUS differs from TTP in severity and lack of neurologic symptoms.

EXAM TIP

The only definitive treatment for hereditary spherocytosis is splenectomy.

EXAM TIP

Diagnostic pentad for TTP: **FAT Rn**
Fever
Anemia
Thrombocytopenia
Renal dysfunction
n eurologic dysfunction

Description

The spleen is an immunologic organ without distinct lobes or segments that weighs about 100–175 g. It is responsible for the removal of old red blood cells and bacteria from the blood circulation.

Anatomic Boundaries

Left upper quadrant (LUQ) of the abdomen, between the 8th and 11th ribs (see Figure 16-1).

- Superior: Left diaphragm leaf.
- Inferior: Colon, splenic flexure, and phrenocolic ligament.
- Medial: Pancreas (tail) and stomach.
- Lateral: Rib cage.
- Anterior: Rib cage, stomach.
- Posterior: Rib cage.

Splenectomy

See Table 16-1 for indications.

Types

- Laparoscopic
- Open

Complications

- Overwhelming postsplenectomy sepsis.
- Atelectasis (not taking deep breaths due to pain)/pneumonia (due to atelectasis sequestering bacteria).
- Pleural effusion (usually on the left).
- Subphrenic abscess.
- Injury to pancreas (because tail of pancreas "hugs" spleen).
- Postoperative hemorrhage.
- Thrombocytosis—many of the platelets that were sequestered in the spleen are now out in the circulation.

Tumors of the Spleen

Benign

- Hemangioma/lymphangioma
- Hamartomas
- Primary cyst/echinococcal cyst

Malignant

- Either lymphomas or myeloproliferative diseases.
- Rare site for solid tumor metastatic disease.
- A common site for metastases especially in lung and breast. However, it is rarely clinically signif cant and usually an autopsy f nding.

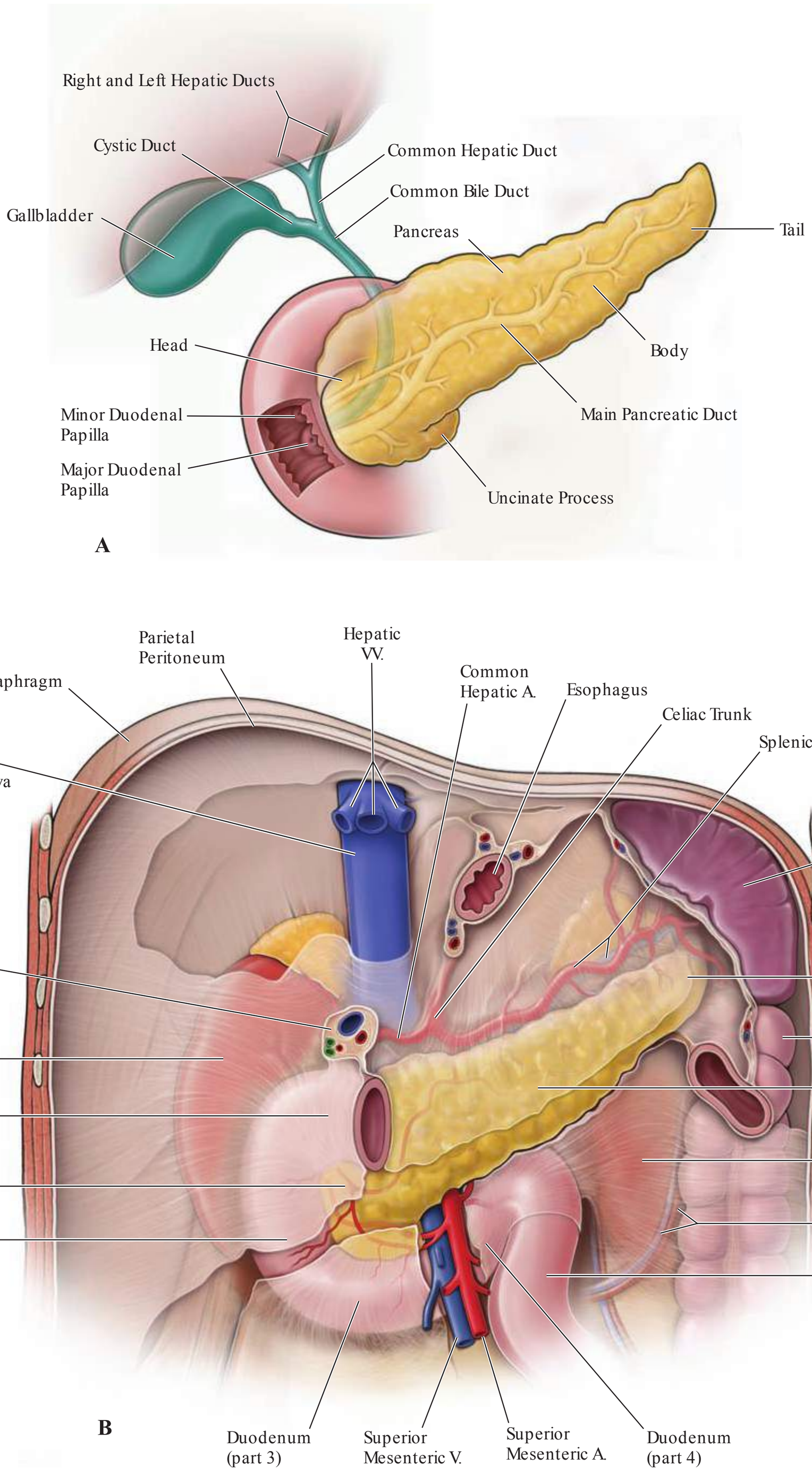


FIGURE 16-1. The spleen and its relationships. (Reproduced, with permission, from Morton DA, Foreman KB, Albertino KH. The Big Picture: Gross Anatomy. New York, NY: McGraw-Hill Education; 2011. Figure 9-3.)

TABLE 16-1. Indications for and Expected Response to Splenectomy in Various Diseases and Conditions

Disease/Condition	Indications for Splenectomy	Response to Splenectomy
Hereditary spherocytosis	Hemolytic anemia, recurrent transfusions, intractable leg ulcers	Improves or eliminates anemia
Hereditary elliptocytosis	Limited role for splenectomy	—
Pyruvate kinase deficiency	Only in severe cases, recurrent transfusions	Decreased transfusion requirement, palliative only
Glucose-6-phosphate dehydrogenase deficiency	None	—
Warm-antibody autoimmune hemolytic anemia	Failure of medical (steroid) therapy	60–80% response rate, recurrences common
Sickle cell disease	History of acute sequestration crisis, splenic symptoms, or infarction (consider concomitant cholecystectomy)	Palliative, variable response
Thalassemia	Excessive transfusion requirements, symptomatic splenomegaly, or infarction	Diminished transfusion requirements, relief of symptoms
Acute myeloid leukemia (AML)	Intolerable symptomatic splenomegaly	Relief of abdominal pain and early satiety
Chronic myeloid leukemia	Symptomatic splenomegaly	Relief of abdominal pain and early satiety
Chronic myelomonocytic leukemia	Symptomatic splenomegaly	Relief of abdominal pain and early satiety
Essential thrombocythemia	Only for advanced disease (i.e., transformation to myeloid metaplasia or AML) with severe symptomatic splenomegaly	Relief of abdominal pain and early satiety
Polycythemia vera	Only for advanced disease (i.e., transformation to myeloid metaplasia or AML) with severe symptomatic splenomegaly	Relief of abdominal pain and early satiety
Myelofibrosis (agnogenic myeloid metaplasia)	Severe symptomatic splenomegaly	76% clinical response at 1 year, high risk of hemorrhagic, thrombotic, and infectious complications (26%)
Chronic lymphocytic leukemia	Cytopenias and anemia	75% response rate
Hodgkin's disease	Surgical staging in selected cases	—
Non-Hodgkin's lymphoma	Cytopenias, symptomatic splenomegaly	Improved complete blood count values, relief of symptoms
Idiopathic thrombocytopenic purpura	Failure of medical therapy, recurrent disease	75–85% rate of long-term response

(continues)

TABLE 16-1. Indications for and expected Response to Splenectomy in Various Diseases and Conditions (continued)

Disease/Condition	Indications for Splenectomy	Response to Splenectomy
Thrombotic thrombocytopenic purpura	Excessive plasma exchange requirement	Typically curative
Abscesses of the spleen	Therapy of choice	Curative
Symptomatic parasitic cysts	Therapy of choice	Curative; exercise caution not to spill cyst contents
Symptomatic nonparasitic cysts	Partial splenectomy for small cysts; unroofing for large cysts	Curative
Gaucher's disease	Hypersplenism	Improves cytopenias; does not correct underlying disease
Niemann–Pick disease		

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Table 34-1.

Splenic Injury

signs and symptoms

- History: Check for preexisting diseases that cause splenomegaly (these patients are more vulnerable to splenic injury), details of injury mechanism.
- Exam: Look for peritoneal irritation, Kehr's sign, left-sided lower rib fractures, external signs of injury.

Treatment

Initial

- Airway, breathing, circulation (ABCs).
- Patients who are stable or who stabilize with fluid resuscitation may be considered for conservative management.
- Further diagnostic tools:
  - Computed tomographic (CT) scan: Able to define injury precisely.
  - Ultrasound (US): May be used for initial assessment to detect hemoperitoneum as a part of focused abdominal sonography for trauma (FAST) exam.
  - Diagnostic peritoneal lavage (DPL): Not specific for splenic injury but will show hemoperitoneum.
  - Angiogram: May be able to use therapeutically in the stable patient (embolization of CT-identified injury).

Definitive

- Nonoperative management criteria:
  - Stable.
  - Injury grade I or II (see Table 16-2).
  - No evidence of injury to other intra-abdominal organs.
  - Consists of bed rest, nasogastric tube (NGT) decompression, monitored setting, serial exam, and hematocrits.

WARD TIP

Transfusing platelets in TTP is thought to "fuel the fire" and exacerbate consumption of platelets and clotting factors, resulting in more thrombi in the microvasculature.

Plasmapheresis is the treatment of choice.

EXAM TIP

The spleen is the most commonly injured organ in blunt abdominal trauma, and trauma is the most common reason for splenectomy.

EXAM TIP

Causes of splenic rupture:

- Trauma: Rib fractures on the left (especially the 9th and 10th, which make up 20% of cases).
- Spontaneous rupture (associated with mononucleosis).

EXAM TIP

Thirty percent of patients with splenic injury will present with hypotensive shock due to hemorrhage.

WARD TIP

Radiographic signs of splenic injury:

- CT: Low-density mass or intrasplenic accumulation of contrast.
- US: Perisplenic fluid, enlarged spleen, irregular borders, abnormal position, increase in size over time.

EXAM TIP

Patients with a vascular blush on CT scan are likely to fail nonoperative management.

TABLE 16-2. AAST Spleen Injury Scale (1994 Revision)

Grade	Injury type	Description of injury
I	Hematoma	Subcapsular, nonexpanding, < 10% surface area
	Laceration	Capsular tear, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, 10–50% surface area; intraparenchymal, nonexpanding, < 5 cm in diameter
	Laceration	Capsular tear, 1–3 cm parenchymal depth that does not involve a trabecular vessel
III	Hematoma	Subcapsular, > 50% surface area or expanding; ruptured subcapsular hematoma with active bleeding; parenchymal hematoma ≥ 5 cm or expanding
	Laceration	> 3 cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (> 25% of spleen)
V	Laceration	Completely shattered spleen
	Vascular	Hilar vascular injury with devascularized spleen

Reproduced, with permission, from Mattox KL, Moore EE, Feliciano DV. Trauma. 7th ed. New York, NY: McGraw-Hill Education; 2013: 568. Table 30-1.

- Operative management indications:
  - Signs and symptoms of ongoing hemorrhage.
  - Injury ≥ grade III.
  - Failure of nonoperative management.
- Exploratory laparotomy:
  - Perform splenectomy if the spleen is the primary source of exsanguinating hemorrhage.
  - If not, pack the area and search for other, more life-threatening injuries; address those first.
  - Subsequently, return to inspection of spleen. Mobilize fully unless the only injury is a minor nonbleeding one.
- Capsular bleeding and most grade II injuries: Apply direct pressure ± topical hemostatic agent.
- Persistently bleeding grade II or III injuries: Suture lacerations.
- Multiple injuries: Consider mesh splenorrhaphy for splenic preservation (especially in children).
- Complex fractures: Perform anatomic resection if possible, based on demarcation after segmental artery ligation.
- Perform splenectomy (see indications).

Splenic Abscess

Causes

- Sepsis seeding
- Infection from adjacent structures

- Trauma
- Hematoma
- IV drug use

signs and symptoms

- Fever, chills.
- LUQ tenderness and guarding.
- Spleen may or may not be palpable.

diagnosis

- US: Enlarged spleen with areas of lucency contained within.
- CT: Abscess will show lower attenuation than surrounding spleen parenchyma. Defines abscess better than US.

Treatment

- Splenectomy for most cases.
- Percutaneous drainage for a large, solitary juxtacapsular abscess.

Complications

- Spontaneous rupture
- Peritonitis
- Sepsis

WARD TIP

Patients who fail nonoperative management usually do so within 48–72 hours.
Delayed bleeding can occur upto several weeks after the injury.

WARD TIP

Pneumococcal, Haemophilus influenzae, and Neisseria meningitidis vaccines will be needed for patients undergoing splenectomy. It may be given 2 weeks postoperatively, or on the day of hospital discharge.

HIGH-YIELD FACTS IN

The Breast

Basic Anatomy 266
- Blood Supply 266
- lymphatic drainage 267
- nerves 267

Boundaries for Mastectomy 268

Boundaries for Axillary Dissection 268

Initial Evaluation of Patients with Possible Breast Disease 268

Evaluation of a Palpable Breast Mass 269
- approach 269
- differential diagnosis 269

Infectious/Inflammatory Conditions 270
- mastitis 270
- hidradenitis Suppurativa 270
- fat necrosis 270

Benign Disease 271
- fibroadenoma 271
- mondor's disease 272
- fibrocystic changes 272
- mammary duct ectasia (plasma cell mastitis) 272
- phyllodes tumor (cystosarcoma phyllodes) 273
- intraductal papilloma 273
- Sclerosing adenosis 273
- gynecomastia 273

Nipple Discharge 273

Premalignant Disease 274

Malignant Tumors 274
- infiltrating ductal carcinoma 274
- infiltrating lobular carcinoma 274
- paget's disease (of the nipple) 274
- inflammatory carcinoma 274

Breast Cancer 275
- epidemiology 275
- risk factors 276
- genetic predisposition 276
- Screening recommendations (from the american cancer Society) 276
- diagnosis and treatment 277
- treatment decisions 278
- types of operations 278
- hormonal therapy: tamoxifen 280
- chemotherapy 281
- reconstruction 281
- recurrence 281
- metastasis 282
- prognosis 282
- breast cancer in pregnant and lactating Women 282
- breast cancer in males 282

Basic Anatomy

WARD TIP

Skin dimpling and retraction of nipple in breast cancer is due to traction on Cooper's ligaments.

The breast is a modified sebaceous gland composed of glandular, fibrous, and adipose tissue (see Figure 17-1).

- Lies within layers of superficial pectoral fascia, anterior to the deep fascia of pectoralis major.
- Each mammary gland consists of approximately 15–20 lobules, each of which has a lactiferous duct that opens on the areola.
- Structural support provided by ligaments that extend from the deep pectoral fascia to the superficial dermal fascia, called Cooper's ligaments.
- Breast tissue frequently extends into axilla as the **axillary tail of Spence.**
- Is partitioned into quadrants by vertical and horizontal lines across the nipple: Upper inner quadrant (UIQ), lower inner quadrant (LIQ), upper outer quadrant (UOQ), and lower outer quadrant (LOQ).
- **Breast Development:**
  - Derived from ectoderm milk streak
  - Estrogen: duct development
  - Progesterone: lobular development

Blood Supply

- Arterial: Axillary artery via the lateral thoracic and thoracoacromial branches, internal mammary artery via its perforating branches, and adjacent intercostal arteries.

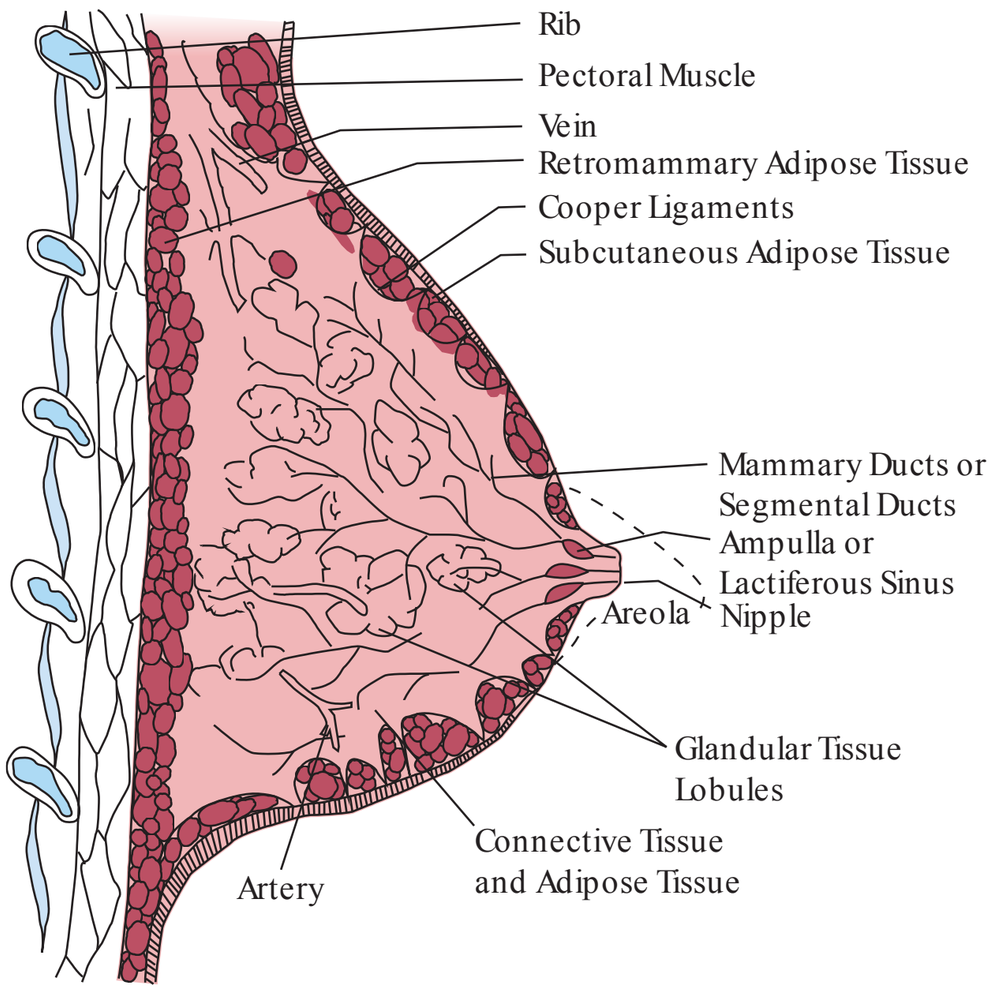


FIGURE 17-1. Cut-away diagram of a mature resting breast. The breast lies cushioned in fat between the overlying skin and the pectoralis major muscle. Both the skin and the retromammary space under the breast are rich with lymphatic channels. Cooper's ligaments, the suspensory ligaments of the breast, fuse with the overlying superficial fascia just under the dermis, coalesce as the interlobular fascia in the breast parenchyma, and then join with the deep fascia of breast over the pectoralis muscle. The system of ducts in the breast is configured like an inverted tree, with the largest ducts just under the nipple and successively smaller ducts in the periphery. After several branching generations, small ducts at the periphery enter the breast lobule, which is the milk-forming glandular unit of the breast. (Reproduced, with permission, from Peart O. Lange Q&A: Mammography Examination. 2nd ed. New York: McGraw-Hill; 2009: 124.)

- Venous: Follows arterial supply; axillary, internal mammary, and intercostal veins; axillary vein responsible for majority of venous drainage.

lymphatic drainage

See Figure 17-2.

- Level I: Lateral to lateral border of pectoralis minor.
- Level II: Deep to pectoralis minor.
- Level III: Medial to medial border of pectoralis minor.
- **Rotter's nodes** lie between the pectoralis major and pectoralis minor muscles.
- Lymphatic drainage from nipple, areola, and lobules all drain in a subareolar lymphatic plexus.
- Ninety-seven percent drain to the axillary lymph nodes
- Two percent drain to the internal mammary nodes
- Any quadrant can drain into the internal mammary nodes

nerves

See Table 17-1.

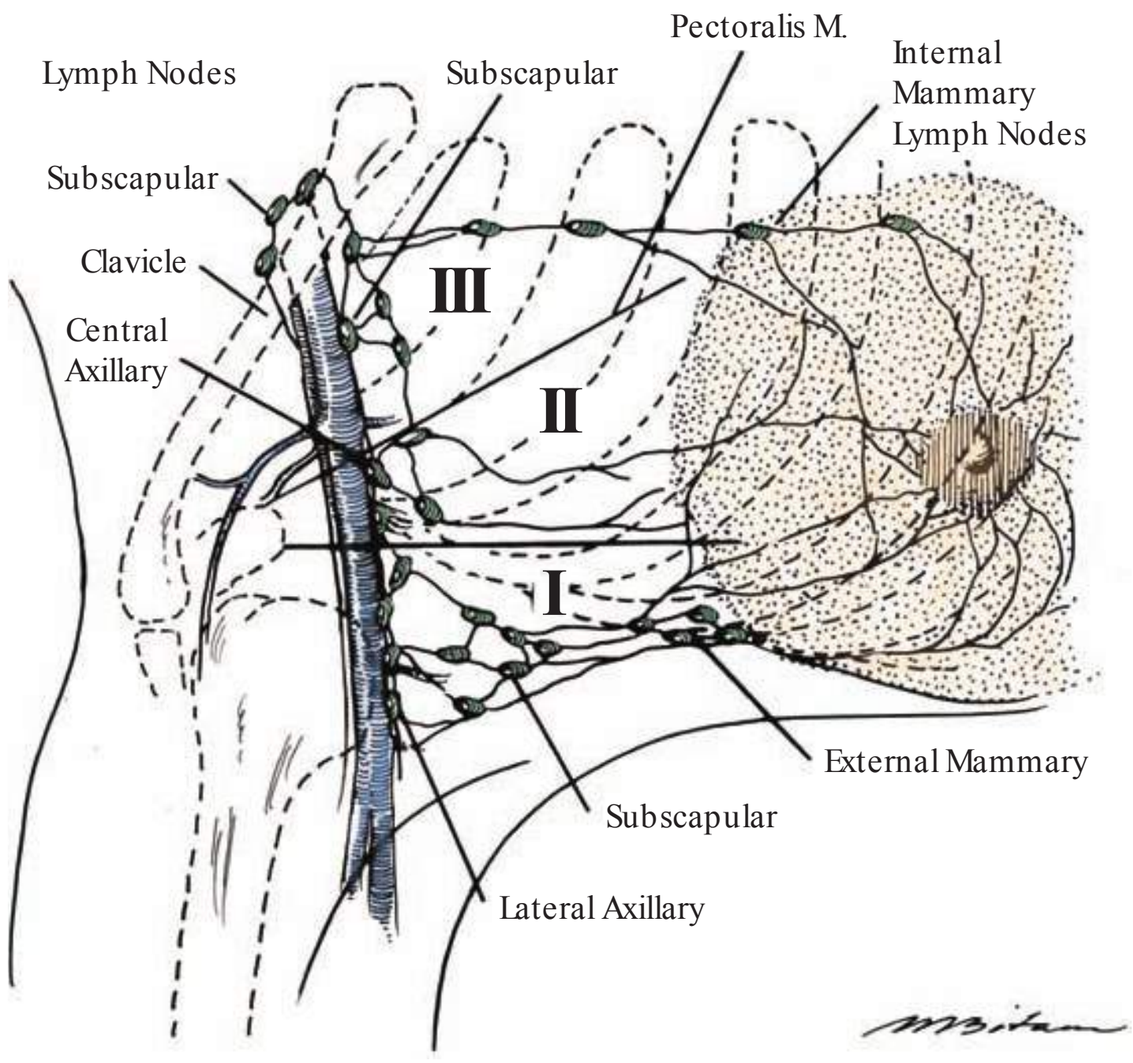


FIGURE 17-2. Contents of the axilla. In this figure there are five named and contiguous groupings of lymph nodes in the full axilla. Complete axillary dissection, as done in the historical radical mastectomy, removes all these nodes. However, note that the subclavicular nodes in the axilla are continuous with the supraclavicular nodes in the neck and nodes between the pectoralis major and minor muscles, named the interpectoral nodes or Rotter's lymph nodes. The internal mammary nodes probably drain independently from the breast. The sentinel lymph node, located in modern sentinel biopsy, is functionally the first and lowest node in the axillary chain. Anatomically, the sentinel lymph node is usually found in the external mammary group. (Reproduced from Zollinger RM Jr, Zollinger RM Sr. Zollinger's Atlas of Surgical Operations. 8th ed. New York, NY: McGraw-Hill; 2003: 397.)

WARD TIP

Venous drainage is largely responsible for metastases to the spine through **paravertebral plexus of Batson.**

WARD TIP

Lymph node involvement by tumor tends to progress from level I to III. The higher the level, the worse the prognosis.

OR TIP

The medial pectoral nerve is actually lateral to the lateral pectoral nerve. The nerves are named according to their origin from the brachial plexus, not by their relation to one another on the chest wall.

OR TIP

The anesthesiologist should not paralyze the patient at surgery because the major nerves (long thoracic and thoracodorsal) are identified by observing muscle contraction when stimulating them with a forceps.

TABLE 17-1. Neural Structures Encountered During Major Breast Surgery

Nerves	Muscle(s)/Area Supplied	Functional deficit if injured
Long thoracic nerve (of Bell)	Serratus anterior	Winging of scapula
Thoracodorsal nerve	Latissimus dorsi	Cannot push oneself up from a sitting position, weak upper extremity adduction
Medial and lateral pectoral nerves	Pectoralis major and minor	Weakness of pectoralis muscles
Intercostobrachial nerve	Crosses axilla transversely to supply inner aspect of arm	Area of anesthesia on inner aspect of arm

Boundaries for Mastectomy

- Superior: Clavicle
- Inferior: Inframammary fold
- Medial: Sternum
- Lateral: Latissimus dorsi

WARD TIP

Perform breast self-examination at same time each month (1 week after menstrual period is ideal).

Boundaries for Axillary Dissection

See Figure 17-2.

- Superior: Axillary vein.
- Posterior: Long thoracic nerve.
- Medial: Either lateral to, underneath, or medial to the medial border of the pectoralis minor muscle, depending on the level of nodes taken.
- Lateral: Latissimus dorsi muscle.

EXAM TIP

Typical scenario: A female with one or more risk factors for breast cancer presents with a mass in the upper outer quadrant of the breast. Think: She's at risk for cancer, and 50% of breast cancers occur in the upper outer quadrant. Therefore, the mass is likely to be malignant.

Initial Evaluation of Patients with Possible Breast Disease

- Complete medical history, including risk factors for breast cancer (see next page). Be sure to inquire about any history of nipple discharge or any changes in the size, shape, symmetry, or contour of the breasts.
- Physical examination (see Figure 17-3).
- Inspection: Note color, symmetry, size, shape, and contour, and check for dimpling, erythema, edema, or thickening of skin with a porous appearance (**peau d'orange**).
- Palpation: Palpate all four quadrants and the nipple–areolar complex for any discharge.
- Axillary nodes are palpated along the lateral border of anterior and posterior axillary fold, the medial and lateral wall of the axilla and the apex of the axilla.

WARD TIP

Palpable breast masses should have a core needle biopsy (as opposed to an FNA)

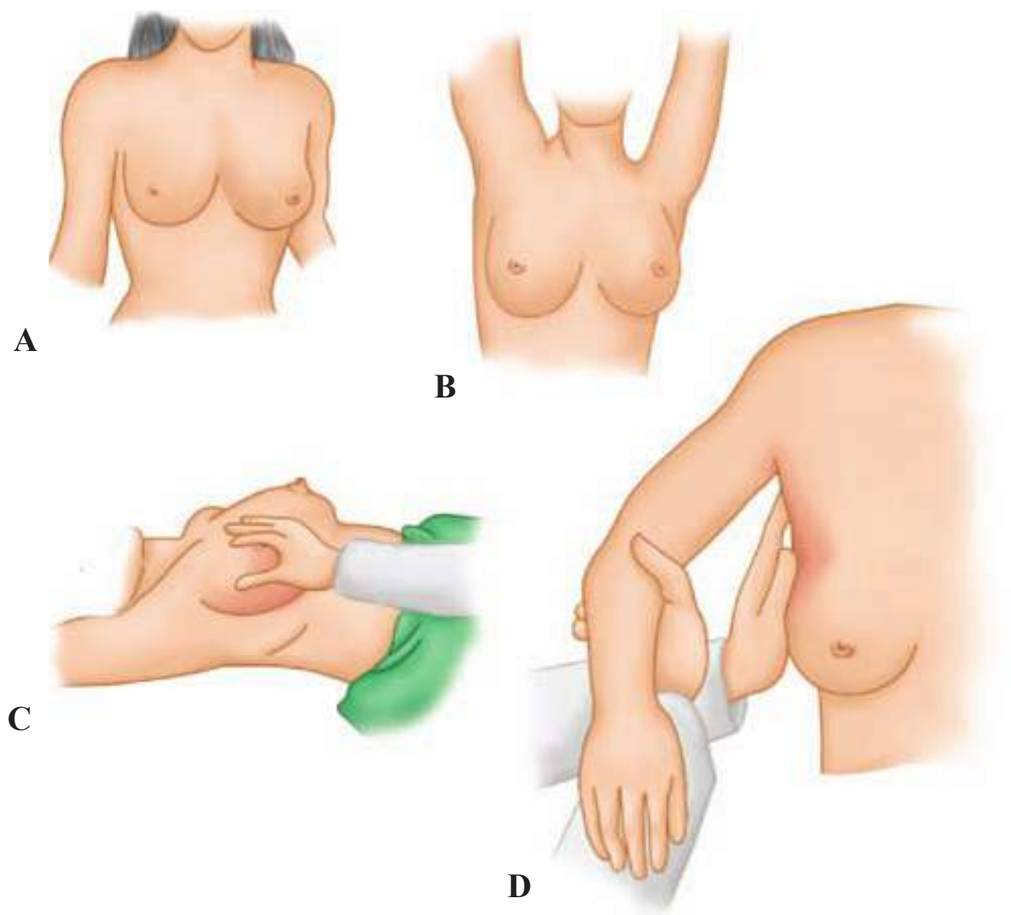


FIGURE 17-3. examination of the breast. A. Inspection of the breast with arms at sides. **B.** Inspection of the breast with arms raised. **C.** Palpation of the breast with the patient supine. **D.** Palpation of the axilla. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 475.)

Evaluation of a Palpable Breast Mass

approach

See Figures 17-4 and 17-5. If age < 30, serial physical examination with observation for 2–4 weeks or until next menstrual period is an option.

differential diagnoSiS

- Infectious/inflammatory: Mastitis, fat necrosis, Mondor's disease.
- Benign lesions: Fibroadenoma, fibrocystic changes, mammary duct ectasia, cystosarcoma phyllodes (occasionally malignant), intraductal papilloma, gynecomastia.

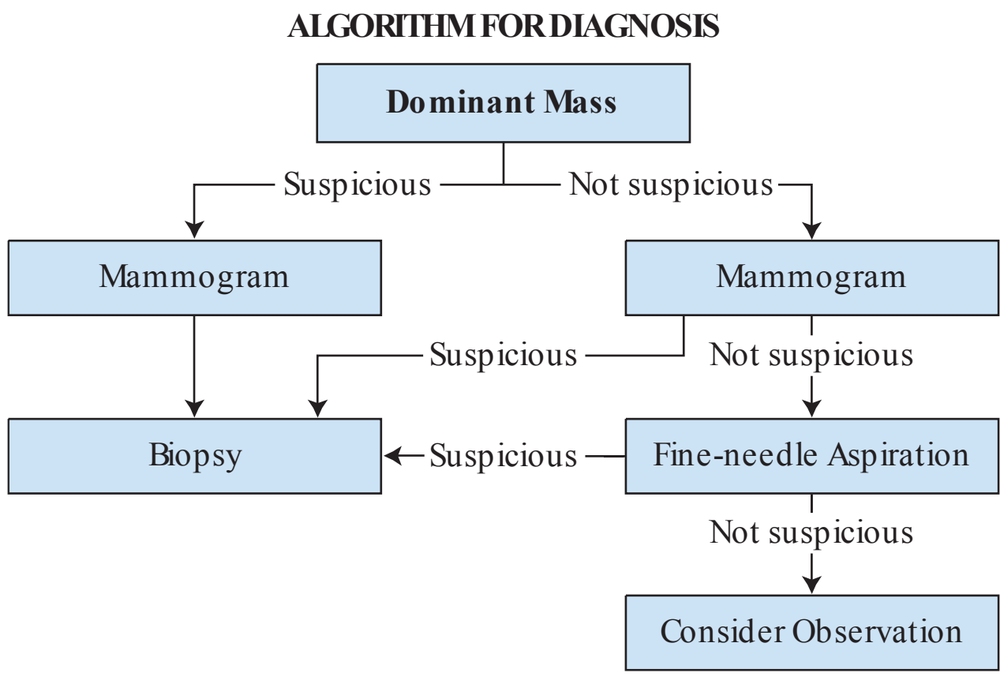


FIGURE 17-4. algorithm for diagnosis of any breast mass (triple method—physical exam, radiology, and tissue diagnosis). (Reproduced, with permission, from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York: McGraw-Hill; 2008: 518.)

WARD TIP

All persistent breast masses require evaluation.

WARD TIP

Only presentations nonsuspicious of cancer:

- Lactating woman with focal erythematous warm swelling.
- Cyclical changing mass in young woman with clear aspirate.

Everything else is extensively worked up.

EXAM TIP

Typical scenario: A 42-year-old woman presents with an undiagnosed breast mass. Think: Evaluate without delay. Observation is not an option if age > 30.

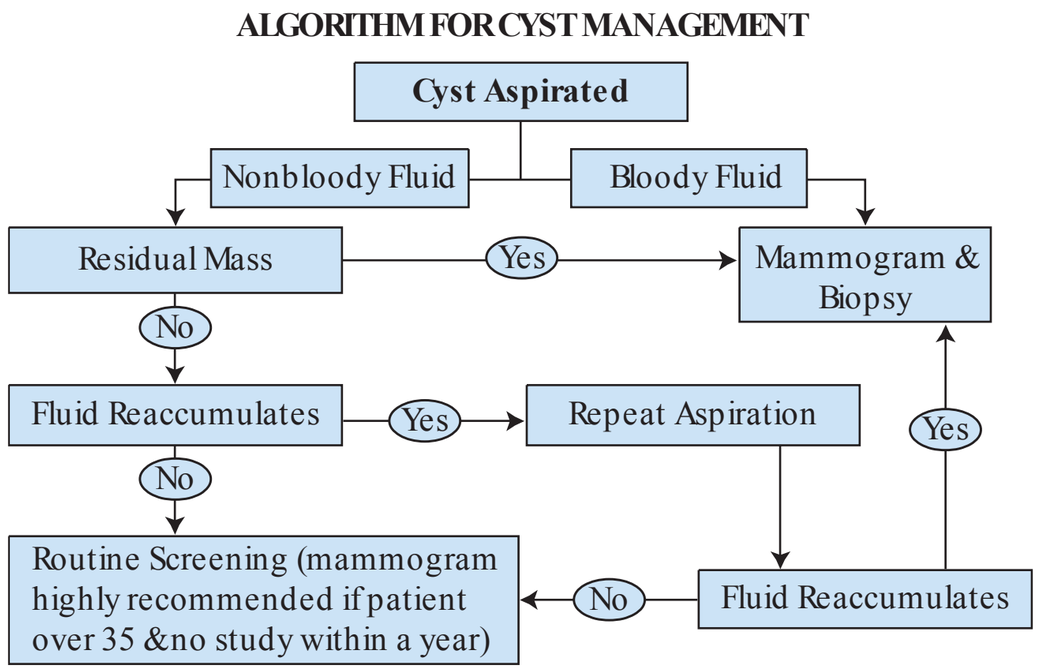


FIGURE 17-5. Algorithm for diagnosis of any breast cyst. (Reproduced, with permission, from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York: McGraw-Hill; 2008: 518.)

- Premalignant disease: Ductal carcinoma in situ (DCIS), lobular carcinoma in situ (LCIS).
- Malignant tumors: Infiltrating ductal, infiltrating lobular, and inflammatory carcinoma; Paget's disease; and other less common histologic types of breast cancer.

EXAM TIP

Typical scenario: A female presents complaining of nipple pain during breast-feeding with focal erythema and warmth of breast on physical exam. Think: Mastitis ± breast abscess. Workup may include ultrasound of the breast, and possible incision and drainage if fluctuance (abscess) is present.

WARD TIP

If a nonlactating women presents with similar picture, consider inflammatory carcinoma.

Infectious/Inflammatory Conditions

mastitis

- Usual etiologic agent: Staphylococcus aureus or Streptococcus spp.
- Most commonly occurs during early weeks of breast-feeding.
- **Physical exam:** Focal tenderness with erythema and warmth of overlying skin, fluctuant mass occasionally palpable.
- **Diagnosis:** Ultrasound can be used to localize an abscess; if abscess is present, aspirate fluid for Gram stain and culture.
- **Treatment:** Prevent engorgement of breast. Continue breast-feeding and recommend use of breast pump as an alternative.
- Cellulitis: Wound care and antibiotics.
- Abscess: Incision and drainage followed by antibiotics.

hidradenitis suppurativa

- A chronic inflammatory condition of the accessory areolar glands of Montgomery; also affects the axilla.
- Women with acne are predisposed to develop hidradenitis.
- May mimic other diseases (Paget's, invasive carcinoma).
- Skin may be involved contiguously or multifocally.
- **Treatment:** Antibiotics with incision and drainage.
- Rarely, skin loss may require skin grafts.

WARD TIP

Typical scenario: A 25-year-old female presents with a painful breast mass several weeks after sustaining breast trauma by a seat belt in a car accident. Think: The most common cause of a persistent breast mass after trauma is fat necrosis.

fat necrosis

- **Presentation:** Firm, irregular mass of varying tenderness.
- History of local trauma elicited in 50% of patients.
- Predisposing factors: Chest wall or breast trauma, previous radiation.

- **Physical exam:** Irregular mass without discrete borders that may or may not be tender; later, collagenous scars predominate.
- Often indistinguishable from carcinoma by clinical exam or mammography.
- **Diagnosis and treatment:** Excisional biopsy with pathologic evaluation for carcinoma.

Benign Disease

See Table 17-2.

fibroadenoma

- **Definition:** Fibrous stroma surrounds ductlike epithelium and forms a benign tumor that is grossly smooth, white, and well circumscribed.
- **Risk factors:** More common in black women than in white women. Most common breast lesion in adolescents and young females.
- **Incidence:** Typically occurs in late teens to early 30s; estrogen sensitive (increased tenderness during pregnancy).
- **Signs and symptoms:** Smooth, discrete, circular, mobile mass.
- **Diagnosis:** FNA.

TABLE 17-2. ANDI Classification of Benign Breast Disorders

	NORMAL	DISORDER	DISEASE
Early reproductive years (age 15–25 years)	Lobular development Stromal development Nipple eversion	Fibroadenoma Adolescent hypertrophy Nipple inversion	Giant fibroadenoma Gigantomastia Subareolar abscess Mammary duct fistula
Later reproductive years (age 25–40 years)	Cyclical changes of menstruation Epithelial hyperplasia of pregnancy	Cyclical mastalgia Nodularity Bloody nipple discharge	Incapacitating mastalgia
Involution (age 35–55 years)	Lobular involution Duct involution Dilatation Sclerosis Epithelial turnover	Macrocysts Sclerosing lesions Duct ectasia Nipple retraction Epithelial hyperplasia	— Periductal mastitis — Epithelial hyperplasia with atypia

ANDI = aberrations of normal development and involution.

Reproduced, with permission, from Hughes LE: Aberrations of normal development and involution (ANDI): A concept of benign breast disorders based on pathogenesis. In: Mansel RE, et al., eds. Hughes, Mansel & Webster's Benign Disorders and Diseases of the Breast. London: Saunders; 2009. Copyright Elsevier.

WARD TIP

Typical scenario: A 20-year-old female presents with a well-circumscribed mass in her left breast. It is mobile, nontender, and has defined borders on physical exam. Think: Fibroadenoma until proven otherwise.

WARD TIP

Mondor's disease most commonly develops along the course of a single vein.

WARD TIP

Typical scenario: A female presents complaining of acute pain in her axilla and lateral chest wall, and a tender cord is identified on physical exam. Think: Mondor's disease versus chest wall infection. Confirm with ultrasound.

WARD TIP

Ten percent of all women develop clinically apparent fibrocystic changes.

WARD TIP

Typical scenario: A 35-year-old female presents with a straw-colored nipple discharge and bilateral breast tenderness that fluctuates with her menstrual cycle. Think: Fibrocystic changes. Consider a trial of OCPs or NSAIDs.

EXAM TIP

Typical scenario: A 45-year-old female presents with breast pain that does not vary with her menstrual cycle with lumps behind the nipple–areolar complex and a history of a nonbloody nipple discharge. Think: Mammary duct ectasia.

- **Treatment:**
  - If FNA is diagnostic for fibroadenoma, may observe if asymptomatic and size < 2 cm. If > 2 cm, symptomatic offer surgical excision.
  - If FNA is nondiagnostic or not synchronous with imaging, must excise mass.
  - In patients > 40 years old, recommend excising mass to ensure diagnosis. The mass is well encapsulated and can be shelled out easily at surgery.

Mondor's disease

- **Definition:** Superficial thrombophlebitis of lateral thoracic or thoracoepigastric vein.
- **Predisposing factors:** Local trauma, surgery, infection, repetitive movements of upper extremity.
- **Presentation:** Acute pain in axilla or superior aspect of lateral breast.
- **Physical exam:** Palpation of tender cord.
- **Diagnosis:** Confirm with ultrasound.
- **Treatment:**
  - Clear diagnosis by ultrasound: Salicylates, warm compresses, limit motion of affected upper extremity. Usually resolves within 2–6 weeks.
  - If persistent, surgery to divide the vein above and below the site of thrombosis or resect the affected segment.
  - Ultrasound nondiagnostic or an associated mass present: Excisional biopsy.

Fibrocystic changes

- Usually diagnosed in 20s–40s.
- **Presentation:** Breast swelling (often bilateral), tenderness, and/or pain.
- **Physical exam:** Discrete areas of nodularity within fibrous breast tissue.
- **Evaluation:** Serial physical examination with documentation of the fluctuating nature of the symptoms is usually sufficient unless a persistent discrete mass is identified; definitive diagnosis requires aspiration or biopsy with pathologic evaluation.
- Symptoms thought to be of hormonal etiology and tend to fluctuate with the menstrual cycle.
- Associated with a group of characteristic histologic findings, each of which has a variable relative risk for the development of cancer.
- Not associated with an increased risk for breast cancer unless biopsy reveals lobular or ductal hyperplasia with atypia.
- **Treatment:**
  - For cases with a classic history or absence of a persistent mass: Conservative management; options include nonsteroidal anti-inflammatory drugs (NSAIDs), oral contraceptive pills (OCPs), danazol, or tamoxifen; advise patient to avoid products that contain xanthine (e.g., caffeine, tobacco, cola drinks).
  - If single dominant cyst, aspirate fluid; may discard if green or cloudy but must send to cytology and excise cyst if bloody.
  - Atypical ductal or lobular hyperplasia: must be excised.

Mammary duct ectasia (plasma cell mastitis)

- **Definition:** Inflammation and dilation of mammary ducts.
- Most commonly occurs in the perimenopausal years.
- **Presentation:** Noncyclical breast pain with lumps under nipple/areola with or without a nipple discharge.
- **Exam:** Palpable lumps under areola, possible nipple discharge.
- **Diagnosis:** Based on exam; excision biopsy required to rule out cancer.
- **Treatment:** Excision of affected ducts.

phyllodes tumor (cystosarcoma phyllodes)

- A variant of fibroadenoma.
- Characterized as benign, intermediate, or malignant phyllodes. Malignant phyllodes are very rare (< 10%).
- Patients tend to present later than those with fibroadenoma (> 30 years).
- **Characteristics:** Indistinguishable from fibroadenoma by ultrasound or mammogram. The distinction between the two entities can be made on the basis of their histologic features (phyllodes tumors have more mitotic activity).
- **Exam:** Large, freely movable mass with overlying skin changes.
- **Diagnosis:** Definitive diagnosis requires biopsy with pathologic evaluation.
- **Treatment:**
  - Smaller tumors: Wide local excision with at least a 1-cm margin.
  - Larger tumors: Simple mastectomy.
  - No need for sentinel lymph node biopsy.

OR TIP

There is no need for axillary node dissection in cystosarcoma phyllodes, as lymph node metastases do not occur. If malignant, will spread hematogenously.

EXAM TIP

Most common cause of unilateral bloody nipple discharge: Intraductal papilloma.

intraductal papilloma

- **Definition:** A benign local proliferation of ductal epithelial cells.
- **Characteristics:** Unilateral serosanguineous or bloody nipple discharge.
- **Presentation:** Subareolar mass and/or spontaneous nipple discharge.
- **Evaluation:** Radially compress breast to determine which lactiferous duct expresses fluid; mammography.
- **Diagnosis:** Definitive diagnosis by pathologic evaluation of resected specimen.
- **Treatment:** Excise affected duct.

EXAM TIP

Typical scenario: A 35-year-old female presents with a 1-month history of a spontaneous unilateral bloody nipple discharge. Radial compression of the involved breast results in expression of blood at the 12 o'clock position. Think: Intraductal papilloma.

sclerosing adenosis

- A sclerosing disorder causing palpable mass in breast.
- Mimics cancer on palpation, mammogram, and gross pathology.
- Excision and histologic exam is diagnostic.

gynecomastia

- **Definition:** Development of female-like breast tissue in males.
- May be physiologic (neonatal, adolescent, and senescent) or pathologic.
  - Pathologies to consider: may be associated with medication use (spironolactone, cimetidine), inappropriately elevated hormonal levels (check prolactin, estrogen, testosterone). If hormones are elevated, evaluate further cause.
- At least 2 cm of excess subareolar breast tissue is required to make the diagnosis.
- **Treatment:** Treat underlying cause if specific cause identified; if normal physiology is responsible, only surgical excision (subareolar mastectomy) may be effective. Surgery is recommended only for progressive gynecomastia or for cosmetic reasons.

WARD TIP

A 14-year-old male complains of gynecomastia. Wait and watch. Perform surgery only if progressive.

Nipple Discharge

- Most nipple discharge is benign
- Evaluation includes history, breast examination, mammography, + /− ultrasound
- Worrisome characteristics: unilateral, spontaneous, serous

WARD TIP

Causes of gynecomastia:

- Increased estrogen (tumors, endocrine disorders, liver failure, obesity).
- Decreased testosterone (aging, primary or secondary testicular failure, Klinefelter's, renal failure).
- Drugs (e.g., spironolactone).

- Green discharge: if cyclical and nonspontaneous, likely due to fibrocystic breast
- Bloody discharge: if unilateral, most likely due to intraductal papilloma
- Bilateral discharge: if milky, consider intracranial etiology, obtain prolactin level

Premalignant Disease

See Table 17-3.

Malignant Tumors

infiltrating ductal carcinoma

- Most common invasive breast cancer (80% of cases).
- Most common in perimenopausal and postmenopausal women.
- Ductal cells invade stroma in various histologic forms described as scirrhous, medullary, comedo, colloid, papillary, or tubular.
- Metastatic to axilla, bones, lungs, liver, brain.

WARD TIP

Tumors **r** unning and **L**eaping **P**romptly to **B**one:

- **T**hyroid
- **r** enal
- **L**ung
- **P**rostate
- **B**reast

infiltrating lobular carcinoma

- Second most common type of invasive breast cancer (10% of cases).
- Originates from terminal duct cells and, like LCIS, has a high likelihood of being bilateral.
- Presents as an ill-defined thickening of the breast.
- Like LCIS, lacks microcalcifications and is often multicentric.
- Tends to metastasize to the axilla, meninges, and serosal surfaces.

paget's disease (of the nipple)

- Two percent of all invasive breast cancers.
- Usually associated with underlying LCIS or ductal carcinoma extending within the epithelium of main excretory ducts to skin of nipple and areola.
- **Presentation:** Tender, itchy nipple with or without a bloody discharge with or without a subareolar palpable mass.
- **Diagnosis:** Biopsy shows Paget's cells.
- **Treatment:** Usually requires a modified radical mastectomy.

inflammatory carcinoma

- Two to 3% of all invasive breast cancers.
- Most lethal breast cancer.
- Frequently presents as erythema, "peau d'orange," and nipple retraction.
- Inflammatory picture is due to the blockage of the efferent lymphatic ducts causing edema—"peau d'orange."
- Diagnosis: Skin biopsy shows dermal lymphatic invasion seen at pathologic evaluation.
- **Treatment:** Consists of chemotherapy followed by surgery and/or radiation, depending on response to chemotherapy.

WARD TIP

Twenty percent of infiltrating lobular breast carcinoma have simultaneous contralateral breast cancer.

TABLE 17-3. Premalignant Disease

disease	dcis	lcis
Cell of origin	Inner layer of epithelial cells in major ducts	Cells of terminal duct–lobular unit
Definition	Proliferation of ductal cells that spread through the ductal system but lack the ability to invade the basement membrane	A multifocal proliferation of acinar and terminal ductal cells. Is NOT precancerous in and of itself
Age and sex	More than half of cases occur after menopause 5% of male cancer	Vast majority of cases occur prior to menopause Never seen in males
Palpable mass	Sometimes	Never
Diagnosis	Clustered microcalcifications on mammogram, malignant epithelial cells in breast duct on biopsy	Typically a clinically occult lesion; undetectable by mammogram and incidental on biopsy
Lymphatic invasion	< 1%	Rare
Risk of invasive	Considered the anatomic precursor of breast carcinoma. Increased risk in ipsilateral breast, usually same quadrant; infiltrating ductal carcinoma most common histologic type; comedo type has the worst prognosis	Considered as a marker of breast carcinoma. Equally increased risk in either breast, infiltrating ductal carcinoma also most common histologic type (counterintuitive); associated with simultaneous LCIS in the contralateral breast in over half of cases
Treatment	▪ If small (< 2 cm): Lumpectomy with either close follow-up or radiation ▪ If large (> 2 cm): Lumpectomy with 1-cm margins and radiation ▪ If breast diffusely involved: Simple mastectomy	None; bilateral mastectomy an option if patient is high risk

Breast Cancer

epidemiology

- One in eight women will develop breast cancer in their lifetime.
- Second most common cause of cancer death among women overall (lung cancer is number 1).
- Incidence increases with increasing age.
- One percent of breast cancers occur in men.

EXAM TIP

Typical scenario: A 65-year-old female presents with a pruritic, scaly rash of her nipple–areolar complex and a bloody nipple discharge. Think: Paget's disease. Biopsy and pathologic exam required to confirm diagnosis.

WARD TIP

Greater than 75% of patients have axillary metastases at time of diagnosis of inflammatory breast carcinoma, and distant metastases are common.

WARD TIP

Paget's disease of vagina is a similar disease, presenting as a scaly, pruritic rash of the vagina.

EXAM TIP

Typical scenario: A 45-year-old female presents with enlargement of her left breast with nipple retraction, erythema, warmth, and induration. Think: Inflammatory breast carcinoma.

WARD TIP

Fibrocystic changes of the breast alone are not a risk factor for breast cancer.

Risk factors

- Any change that causes increased exposure to estrogen without the protective effects of progesterone.
- Early menarche (< 12).
- Late menopause (> 55).
- Nulliparity or first pregnancy > 30 years.
- White race.
- Old age.
- History of breast cancer in mother or sister (especially if bilateral or premenopausal).
- Genetic predisposition (BRCA1 or BRCA2 positive, Li–Fraumeni syndrome).
- Prior personal history of breast cancer.
- Previous breast biopsy.
- DCIS or LCIS.
- Atypical ductal or lobular hyperplasia.
- Postmenopausal estrogen replacement (unopposed by progesterone).
- Radiation exposure.

WARD TIP

Despite all known risk factors, most women with breast cancer (75%) present without any identifiable risk factors.

Genetic predisposition

- Five to 10% of breast cancers are associated with an inherited mutation.
- p53: A tumor suppressor gene; Li–Fraumeni syndrome results from a p53 mutation.
- Both BRCA1 and BRCA2 function as tumor-suppressor genes, and for each gene, loss of both alleles is required for the initiation of cancer.
  - BRCA1 and BRCA2 both are inherited in an autosomal dominant fashion with varying penetrance.
  - BRCA1: On 17q, also associated with ovarian cancer. Sixty percent lifetime risk of breast cancer.
  - BRCA2: On chromosome 13q, also associated with male breast cancer. Sixty percent lifetime risk of breast cancer.
- Somatic mutation of p53 in 50% and of Rb in 20% of breast cancers.

WARD TIP

Genetic syndromes associated with breast cancer:

- Autosomal dominant:
  - Li–Fraumeni
  - Muir–Torre
  - BRCA1 and BRCA2 Cowden's syndrome
  - Peutz–Jeghers syndrome
- Autosomal recessive:
  - Ataxia-telangiectasia

Screening recommendations (from the american cancer Society)

See Table 17-4. Screening reduces mortality by 30–40%.

Table 17-4. American Cancer Society Recommendations for Breast Cancer Detection in a Symptomatic Women

Age group	Examination	Frequency
20–39	Breast self-examination	Monthly
	Clinical breast examination	Every 3 years
40 and older	Breast self-examination	Monthly
	Clinical breast examination	Annually
	Mammography	Annually

Reproduced, with permission, from Chen MYM, Pope TL, Ott DJ. Basic Radiology. New York: McGraw-Hill; 1996: 123.

diagnoSiS and treatment

Diagnostic Options

- **Mammography:** See Figure 17-6.
  - Identifies 5 cancers/1,000 women.
  - Sensitivity 85–90%.
  - False positive 10%, false negative 6–8%.
  - If cancer is first detected by mammogram, 80% have negative lymph nodes (vs. 45% when detected clinically). Mammography picks up early disease and hence is a good screening test.
    - Suspicious finding: Stellate, speculated mass with associated microcalcifications.
- **Reporting mammogram results (Breast Imaging Reporting and Data System—BIRADS):**
  0: Assessment incomplete
  1: Negative
  2: Benign finding
  3: Probably benign finding
  4: Suspicious for cancer
  5: Highly suspicious for cancer
  6: Known biopsy-proven cancer, treatment pending
- **Ultrasound:**
  - (+ +) No ionizing radiation.
  - (+) Good for identifying cystic disease and can also assist in therapeutic aspiration.
  - (+) Results easily reproducible.
  - (–) Resolution inferior to mammogram. Used as adjunct if concerning mass is found on mammography.
  - (–) Will not identify lesions < 1 cm.
- **FNA (aspiration of tumor cells with small-gauge needle):**
  - Advantages: Low morbidity, cheap, only 1–2% false-positive rate.
  - Disadvantages: False-negative rate up to 10%, requires a skilled pathologist.
- **Needle localization biopsy:** Locates occult cancer in > 90%.

WARD TIP

Start yearly mammograms 10 years before the age at which first-degree relative was diagnosed with breast cancer.

WARD TIP

Mammography is more useful if age > 30 because the large proportion of fibrous tissue ("dense tissue") in younger women's breasts make mammograms more difficult to interpret.

WARD TIP

Benign cysts should not be bloody. A bloody aspirate usually indicates malignancy.

WARD TIP

Five to 10% of palpable masses have a negative mammogram.

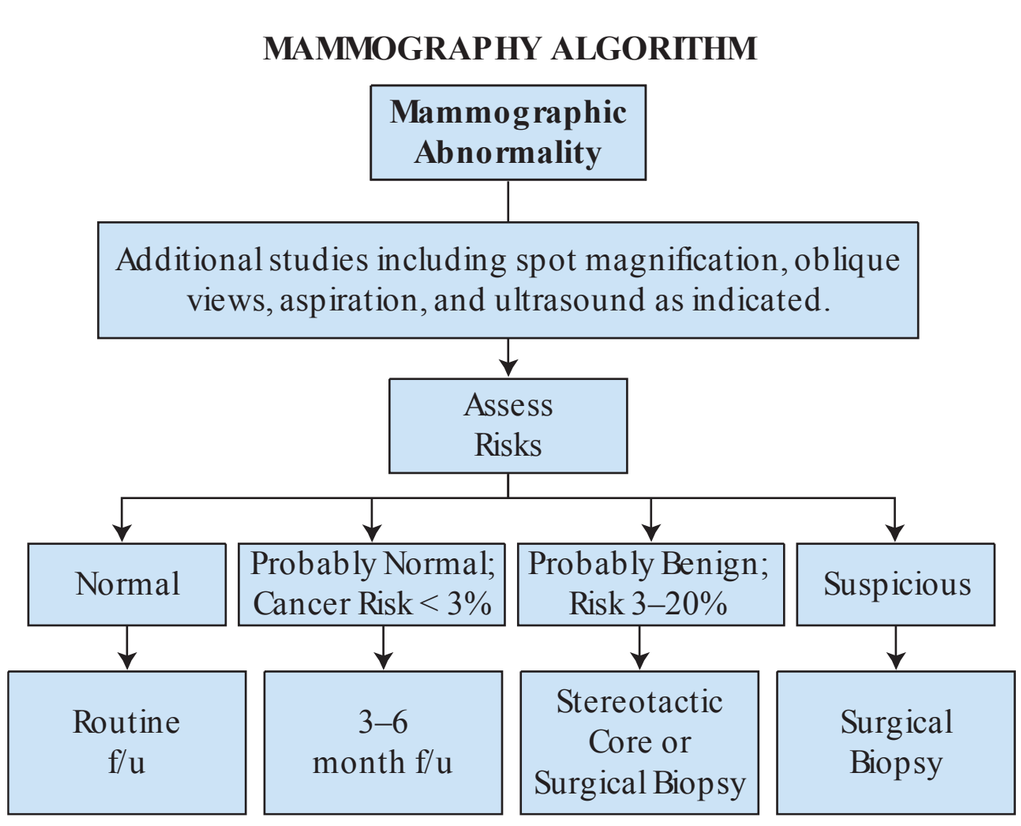


FIGURE 17-6. approaches to abnormalities detected by mammogram. (Reproduced, with permission, from Fauci AS, Braunwald E, Kasper DL, et al. Harrison's Principles of Internal Medicine. 17th ed. New York: McGraw-Hill; 2008: 518.)

- **Core biopsy:** Chance of sampling error.
- **Stereotactic core biopsy:**
  - Fewer complications compared to needle localization biopsy.
  - Less chance of sampling error than core biopsy alone.
  - No breast deformity.

treatment deciSionS

See Table 17-5.

Early Invasive Breast Cancer (Stages I and II)

- For early stages (I and II): Currently, mastectomy with axillary sentinel lymph node biopsy (which evaluates the status of axillary lymph nodes) and breast conservation (lumpectomy, sentinel lymph node biopsy, and adjuvant radiation therapy) are considered equivalent treatments for stage I and II breast cancer (see Table 17-6).
- Adjuvant chemotherapy for early invasive breast cancer is considered for all node-positive cancers, all cancers that are larger than 1 cm in size, and node-negative cancers larger than 0.5 cm in size when blood vessel or lymph vessel invasion, high nuclear grade, high histologic grade, HER2/neu overexpression, and negative hormone receptor status is present (see Table 17-7).
- Tamoxifen (estrogen blocker) therapy is considered for hormone receptor–positive women with cancers that are larger than 1 cm in size.

Locally Advanced Breast Cancer (Stage III)

See Figure 17-7.

- If proceeding with surgery first, would expect mastectomy with axillary lymph node dissection for pathologically confirmed nodal disease.
- Consideration can be made for neoadjuvant chemotherapy, for assessment of tumor responsiveness or to shrink tumor for ease of surgery. At time of surgery, still expect to proceed with axillary lymph node dissection.

Advanced Breast Cancer (Stage IV)

- Systemic and palliative treatment.
- Some patients need mastectomy for palliation and relief from foul discharge and pain.

WARD TIP

Prognosis depends more on stage than on histologic type of breast cancer. Node positivity is the most important factor.

WARD TIP

Lumpectomy with sentinel node biopsy and postoperative radiation is a viable treatment option only in stages I and II. Mastectomy is not required and has no additional survival benefit.

typeS of operationS

- **Simple mastectomy:** Removal of all breast tissue.
- **Modified radical mastectomy:** Resection of all breast tissue and axillary nodes (level I).
- **Radical mastectomy:** Resection of all breast tissue, axillary nodes, and pectoralis major and minor muscles (rarely performed nowadays due to increased morbidity without advantage).
- Breast-conserving surgery:
  - **Lumpectomy and axillary node dissection:** Resection of mass with rim of normal tissue and axillary node dissection—good cosmetic result. Axillary dissection is generally carried out in levels I and II. Zone III is explored only if nodes are palpable.
  - Quadrantectomy and axillary radiotherapy (QUART).
- Breast conservation is considered for all patients with stage I or II cancer because of the important cosmetic advantages. Relative contraindications to breast-conservation therapy include:
  - Prior radiation therapy to the breast or chest wall.
  - Involved surgical margins or unknown margin status following reexcision.

TABLE 17-5. TNM staging for Breast Cancer

PRIMARY TUMOR (T)	
Tx	Primary tumor cannot be assessed
T0	No evidence of tumor
Tis	Carcinoma in situ
DCIS	Ductal carcinoma in situ
LCIS	Lobular carcinoma in situ
T1	< 2 cm
T2	> 2 cm but < 5 cm
T3	> 5 cm
T4	Any size tumor with direct extension to the chest wall and/or to the skin (including inflammatory breast cancer or peu d'orange)

CLINICAL NODES (N)	
Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis to movable ipsilateral level I, II axillary lymph nodes
N2	Metastasis in ipsilateral level I, II axillary lymph nodes that are clinically matted or fixed
N3	Metastasis in ipsilateral infraclavicular or supraclavicular lymph nodes with or without level I, II involvement; or in clinically detected ipsilateral internal mammary lymph nodes with clinically evident level I, II axillary lymph nodes

DISTANT METASTASIS (M)	
M0	No clinical or radiographic evidence of distant metastasis
M1	Distant detectable metastasis as determined by classic clinical and radiographic means and/or histologically proven larger than 0.2 mm

ANATOMIC STAGING			
Stage 0	Tis	N0	M0
Stage IA	T1	N0	M0
Stage IB	T0	N1	M0
	T1	N1	M0
Stage IIA	T0	N1	M0
	T1	N1	M0
	T2	N0	M0
Stage IIB	T2	N1	M0
	T3	N0	M0
Stage IIIA	T0	N2	M0
	T1	N2	M0
	T2	N2	M0
	T3	N1	M0
	T3	N2	M0
Stage IIIB	T4	N0	M0
	T4	N1	M0
	T4	N2	M0
Stage IIIC	Any T	N3	M0
Stage IV	Any T	Any N	M1

TABLE 17-6. Staging System for Breast Cancer

Stage 0	DCIS or LCIS
Stage I	Invasive carcinoma ≤ 2 cm in size (including carcinoma in situ with micro invasion) without nodal involvement and no distant metastases
Stage II	Invasive carcinoma ≤ 5 cm in size with involved but movable axillary nodes and no distant metastases, or a tumor > 5 cm without nodal involvement or distant metastases
Stage III	Breast cancers > 5 cm in size with nodal involvement; or any breast cancer with fixed axillary nodes; or any breast cancer with involvement of the ipsilateral internal mammary lymph nodes; or any breast cancer with skin involvement, pectoral and chest wall fixation, edema, or clinical inflammatory carcinoma, if distant metastases are absent
Stage IV	Any form of breast cancer with distant metastases

TABLE 17-7. Hormone Receptor Status Response to Therapy

Hormone Receptor Status	Response to Hormonal Therapy (Estrogen Blockers or Aromatase Inhibitors)
ER+/PR+	80%
ER–/PR+	45%
ER+/PR–	35%
ER/PR–	10%

- Multicentric disease.
- Scleroderma or other connective-tissue disease.
- Pregnancy

- **Sentinel node biopsy:** Recently developed alternative to complete axillary node dissection:
  - Done only if there are no palpable nodes.
  - Based on the principle that metastatic tumor cells migrate in an orderly fashion to first draining lymph node(s).
  - Lymph nodes are identified on preoperative nuclear scintigraphy and blue dye is injected in the periareolar area.
  - Axilla is opened and inspected for blue and/or "hot" nodes identified by a gamma probe.
  - When sentinel node(s) is positive, an axillary dissection is completed.
  - When sentinel node(s) is negative, an axillary dissection is not performed unless axillary lymphadenopathy is identified.

hormonal therapy: tamoxifen

- Selective estrogen receptor modulator (SERM) that blocks the uptake of estrogen by target tissues.

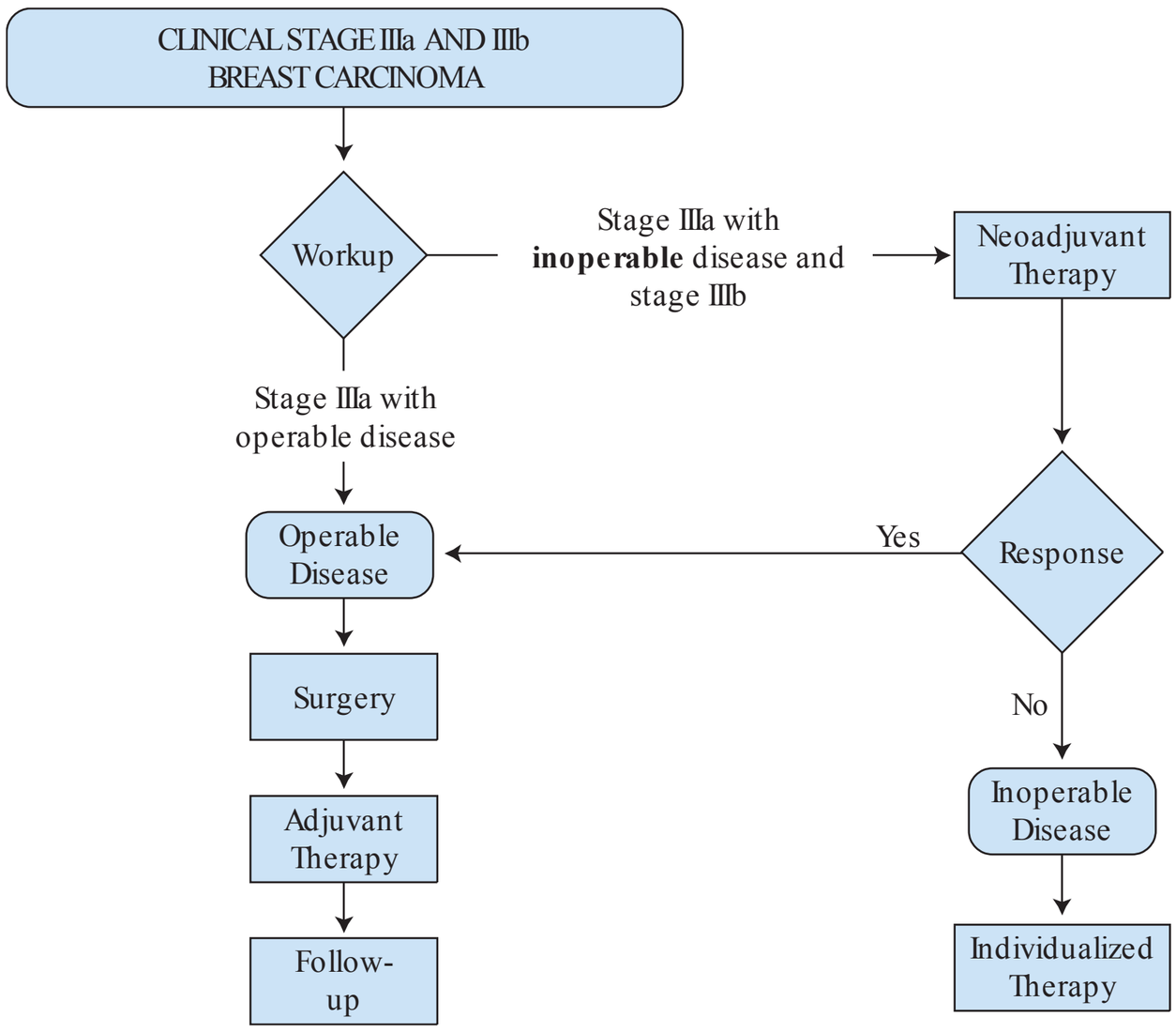


FIGURE 17-7. Treatment pathways for stage IIIa and IIIb breast carcinoma.

- Side effects will simulate menopause: Hot flashes, irregular menses; thromboembolism and increased risk for endometrial cancer due to selective hormone agonist action.
- Therapy of choice for postmenopausal women with positive receptors. Survival benefit for pre- and postmenopausal women, but benefit greater for ER-positive patients.
- May get additional benefit by combining tamoxifen with chemotherapy.



WARD TIP

Raloxifene is another SERM that has been used for breast cancer prevention. It does not increase uterine cancer risk.

chemotherapy

- Any number of regimens are acceptable. Adriamycin, 5-fuorouracil, methotrexate, cyclophosphamide, and paclitaxel (Taxol) are some of the agents used.
- Herceptin is used for HER2/neu-positive patients.

reconStruction

- Done in patients after mastectomy.
- Can use either prosthetic or autologous implants. May also use an expander and implant at a later date.
- Prosthetic implant may be either saline or silicone based.
- Autologous tissue can be either rectus muscle or latissimus dorsi muscle myocutaneous flap.

recurrence

- Five to 10% local recurrence at 10 years.
- Metastases in < 10% of cases.
- Local chest wall recurrence most common within 2–3 years, if at all.

metastasis

- Median survival 2 years.
- Palliative therapy indicated.

prognosis

The 5-year survival rate is:

- Stage I: 94%
- Stage II: 70–85%
- Stage III: 48–52%
- Stage IV: 18%

WARD TIP

Termination of pregnancy is not part of the treatment plan for breast cancer and does not improve survival.

Breast cancer in pregnant and lactating Women

- Three breast cancers are diagnosed per 10,000 pregnancies.
- A fine-needle aspiration should be performed. If it identifies a solid mass, then it should be followed by biopsy.
- Mammography is possible as long as proper shielding is used.
- Radiation is not advisable for the pregnant woman. Thus, for stage I or II cancer, a modified radical mastectomy should be done rather than a lumpectomy with axillary node dissection and postoperative radiation. If diagnosed in the third trimester, only then can lumpectomy and adjuvant radiation be considered. Radiation would be given postpartum.
- If lymph nodes are positive, delay chemotherapy until the second trimester.

WARD TIP

Males with breast cancer often have direct extension to the chest wall at diagnosis.

Breast cancer in males

- Predisposing factors: Klinefelter's syndrome, estrogen therapy, elevated endogenous estrogen, previous irradiation, and trauma.
- Infiltrating ductal carcinoma most common histologic type (men lack breast lobules).
- Diagnosis tends to be late, when the patient presents with a mass, nipple retraction, and skin changes.
- Stage by stage, survival is the same as it is in women. However, more men are diagnosed at a later stage.
- Treatment for early-stage cancer involves a modified radical mastectomy and postoperative radiation.
- Sentinel node biopsy and estrogen receptor status may help to stage disease.

Burns

Burn Injury Basics 284
- American Burn Association criteria for referral to a Burn center 284

Fluid Resuscitation 285
- Adults 285
- Pediatric Patients 285
- monitoring fluid status 285
- increase in fluid requirements 286

Burn Care 286
- daily 286
- definitive therapy 287

Operations 287
- escharotomy 287
- fasciotomy 288
- debridement and skin grafting 288

Infection 288
- treatment 288

Inhalational Injury and Carbon Monoxide Poisoning 289
- Signs and Symptoms 289
- diagnosis 289
- treatment 289
- complications 289

Electrical Injury 289
- definition 289
- mechanism 290
- Signs and Symptoms 290
- treatment 290
- late complications 290

Chemical Injury 290
- mechanism 290
- Severity 290
- Priorities in treatment 291

Nutrition of Burn Patients 291

Burn Scar Cancer 291

WARD TIP

Incidence of burns is higher during winter months.

WARD TIP

Seventy-five percent of burn-related deaths can be attributed to house fires.

EXAM TIP

Typical scenario: A 4-year-old child is brought to your emergency department (ED) with a 14% total body surface area (TBSA) burn, including both second and third degree. It looks as if he had been seated in scalding water. Where should this child be cared for? Think: This patient is under 10 years old, with > 10% TBSA burned, including the perineum and genitalia, and may require special social assistance (due to likelihood of child abuse). He should be transferred to a burn center.

Burn Injury Basics

Most common cause of burn injuries is **scalding** (of which 80% may be treated as outpatient).

American Burn Association criteria for referral to a Burn center

- Partial thickness burns greater than 10% total body surface area (TBSA).
  - Burns that involve the face, hands, feet, genitalia, perineum, or major joints.
  - Third-degree burns in any age group.
  - Electrical burns, including lightning injury.
  - Chemical burns.
  - Inhalation injury.
  - Burn injury in patients with preexisting medical disorders that could complicate management, prolong recovery, or affect mortality.
  - Any patient with burns and concomitant trauma (such as fractures) in which the burn injury poses the greatest risk of morbidity or mortality. In such cases, if the trauma poses the greater immediate risk, the patient may be initially stabilized in a trauma center before being transferred to a burn unit.
  - Burned children in hospitals without qualified personnel or equipment for the care of children.
  - Burn injury in patients who will require special social, emotional, or rehabilitative intervention.

Treatment Outline

1. Airway, breathing, circulation (ABC); intubate patient if indicated. Start fluid resuscitation with 1 L lactated Ringer's (LR) solution bolus in adults, and 20 mL/kg for children in the ED.
2. If carbon monoxide inhalation is suspected, administer 100% oxygen by nonrebreather mask (accelerates dissociation).
3. Assess area of burn (second and third degree).
4. All burn patients need good analgesia (IV morphine). Apply cold saline soaks for analgesia if burns are < 25% BSA (watch for hypothermia).
5. Cover burns with silver sulfadiazine/clean sheet and then warm blanket.
6. Elevate burned areas when possible to minimize edema.
7. Basic laboratories: Arterial blood gas (ABG), complete blood count (CBC), electrolytes, carboxyhemoglobin.
8. Weigh patient.
9. Continue fluid resuscitation as per the Parkland formula.
10. Insert Foley catheter in patients requiring fluid resuscitation or with significant perineal burns.
11. Electrocardiogram (ECG).
12. Nasogastric tube for extensive burns (prone to ileus). Gastrointestinal prophylaxis for Curling's ulcers.

Patients with high-voltage electrical injury require cardiac monitor, as do any intubated or otherwise unstable patients.

Assessment of Extent and severity of Burns

- Consider only second- and third-degree burns when stating %BSA burned.
- The Rule of Nines may be used to estimate the area burned (see Figure 18-1).
- The palm of the patient's hand is roughly equivalent to 1% BSA.

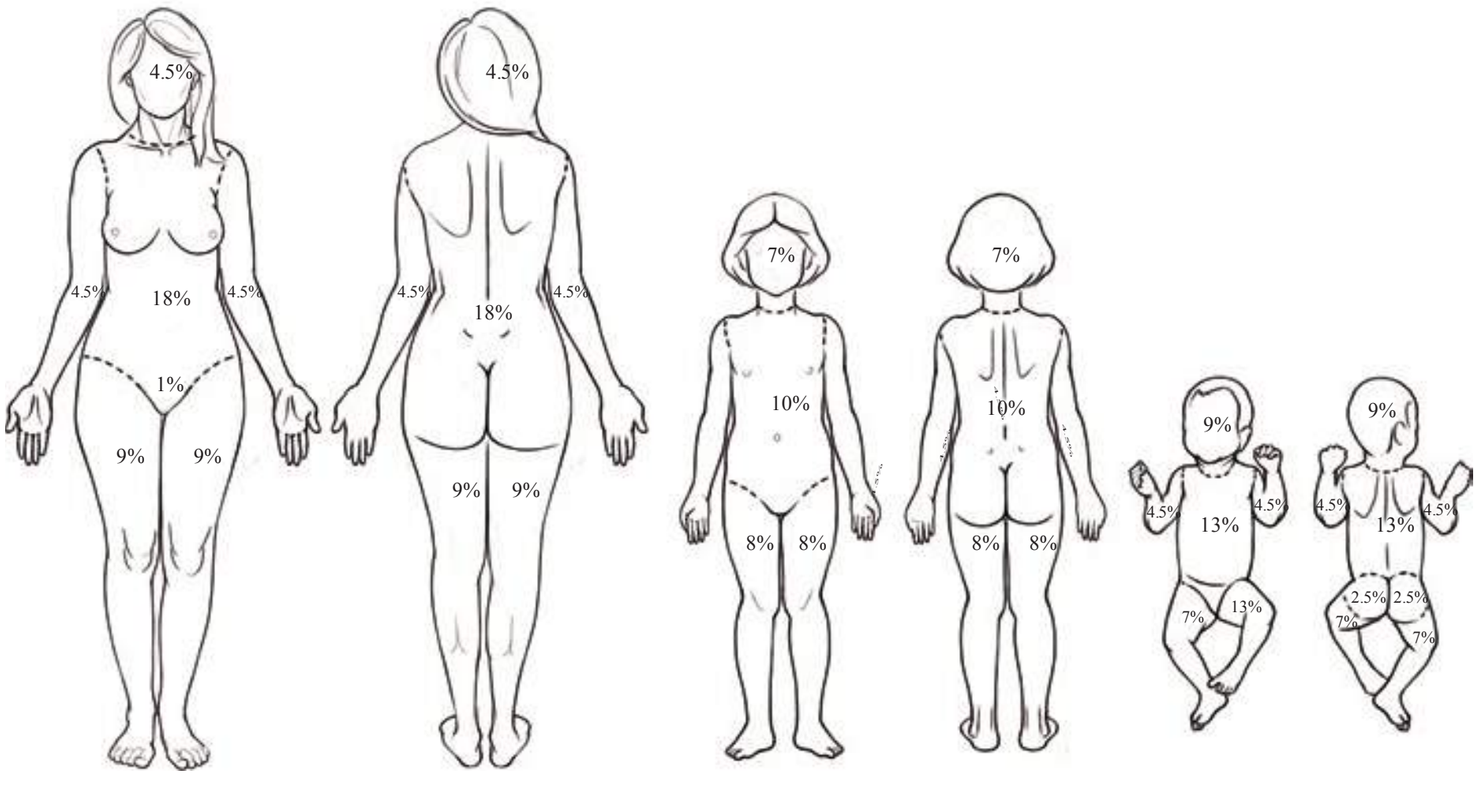


FIGURE 18-1. Rule of nines for estimating body surface area burned in adults, children, and infants.

- Percent BSA burned is used to determine need for fluid resuscitation (see below).
- Patients not requiring admission to any hospital should receive appropriate management and follow-up, including cleansing of burned areas; debridement of loose, nonviable skin; application of topical antimicrobial/nonadherent dressing or of biologic dressing (described below).
- Assess need for escharotomy or fasciotomy (see below).

Deciding Who May Be Treated as an Outpatient

- Most first-degree burns.
- Superficial and intermediate second-degree burns of < 10% BSA (excluding most burns of face, eyes, hands, perineum).
- Patients with acceptable social situations amenable to providing a safe and helpful environment at home.

Fluid Resuscitation

Adults

Parkland Formula

For first 24 hours:

- LR at rate of 4 mL/kg/%BSA burn.
- Give half of 24-hour requirement in first 8 hours from the time of burn, and the remainder over the next 16 hours.

Pediatric Patients

Galveston Formula

$$5{,}000\ \text{mL/m}^2 \text{ burned area} + 1{,}500\ \text{mL/m}^2 \text{ total area}$$

monitoring fluid status

- Resuscitation is adequate when urine output is 30–50 cc/hr in adults and 1 cc/kg/hr in children < 30 kg.

WARD TIP

Everyone involved in the care of the newly arrived burn patient should use sterile technique, even when simply examining the patient. The risk of infection is extremely high with skin loss, since the skin is our biggest immune defense.

EXAM TIP

Typical scenario: An adult male is brought to the ED with second-degree burns on his chest and abdominal wall, anterior right leg, and perineum. What percentage TBSA does he have? Think: Rule of Nines says 18% for anterior torso, 9% for anterior leg, and 1% for perineum = 28%.

EXAM TIP

Typical scenario: How much fluid should a 60-kg female with a 25% TBSA burn receive during the first 24 hours? Think: Parkland formula. At 4 mL/kg/%, 4 × 60 × 25 = 6,000 mL required over the next 24 hours, at a rate of 375 mL/hr for the first 8 hours, and 188 mL/hr for the next 16 hours.

WARD TIP

The risk of infection of burned tissue is increased because the wound is protein rich and moist, and is thus a good culture medium. The neoeschar and lack of vascularity limit antibiotic delivery.

- Adjust fuids when urine output is more than 33% different (in either direction) from recommended over 2–3 hours.
- Monitor daily weights.

increAse in fluid requirements

- High-voltage electrical injury
- Inhalational injury
- Delayed resuscitation
- Intoxicated at time of injury

Burn Care

dAily

- ABC, physiologic resuscitation.
- Cleanse wounds, trim nonviable skin.
- Topical antimicrobial agents for burn wound care (see Table 18-1).
- Daily burn care in shower or tank if possible; otherwise, at bedside.

TABLE 18-1. Topical Antimicrobial Agents for Burn Wound Care

Silver Nitrate	MafeNide acetate	Silver SulfadiaziNe
Active component		
0.5% in aqueous solution	11.1% in water-miscible base	1.0% in water-miscible base
spectrum of antimicrobial activity		
Gram-negative—good	Gram-negative—good	Gram-negative—variable
Gram-positive—good	Gram-positive—good	Gram-positive—good
Yeast—good	Yeast—poor	Yeast—good
Method of wound care		
Occlusive dressings	Exposure	Exposure or single-layer dressings
Advantages		
Painless	Penetrates eschar	Painless
No hypersensitivity reaction	Wound appearance readily monitored	Wound appearance readily monitored when exposure method used
No gram-negative resistance	Joint motion unrestricted	Easily applied
Dressings reduce evaporative heal loss	No gram-negative resistance	Joint motion unrestricted when exposure method used
Greater effectiveness against yeasts		Greater effectiveness against yeast
Disadvantages		
Def cits of sodium, potassium, calcium, and chloride	Painful on partial-thickness burns	Neutropenia and thrombocytopenia
No eschar penetration	Susceptibility to acidosis as a result of carbonic anhydrase inhibition	Hypersensitivity—infrequent
Limitation of joint motion by dressings	Hypersensitivity reactions in 7% of patients	Limited eschar penetration
Methemoglobinemia—rare		
Argyria—rare		
Staining of environment and equipment		

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 213.

definitive therAPy

- For definitive nonexcisional therapy, continue daily cleansing and debridement until granulation tissue is present with minimal or no necrotic debris, at which time a biologic dressing is applied.
- For operative therapy, see Operations section.

Operations

escharotomy

- Areas of concern: Circumferential burns of extremities (including penis) or thorax (see Figure 18-2).
- Indications:
  - Impairment of circulation in extremities
  - Impaired ventilation
- No anesthesia is needed.

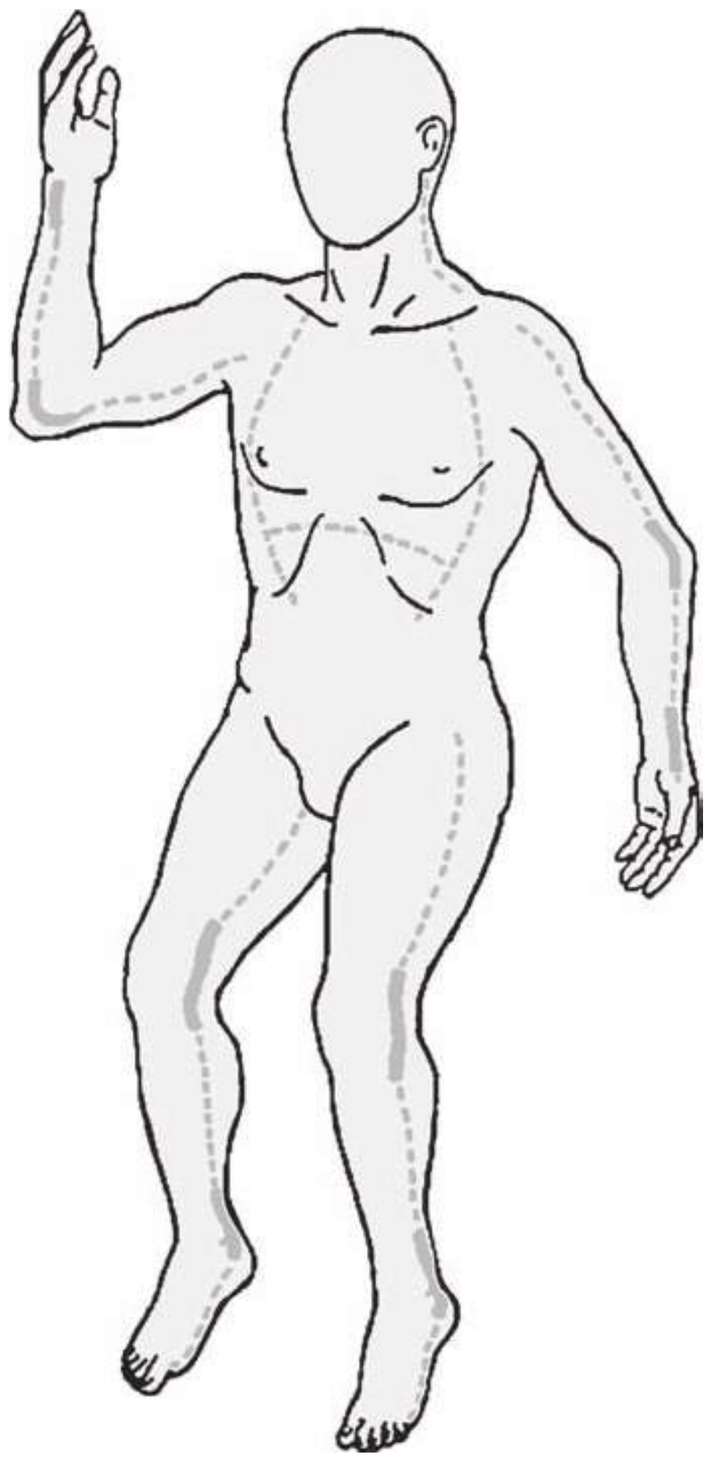


FIGurE 18-2. Locations for escharotomies. The incisions are placed along the mid-medial lines of the extremities and the thorax (dashed lines). The skin is especially tight along major joints, and decompression at these sites must be complete (solid lines). Neck and digital escharotomies are rarely necessary. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 8th ed. New York: McGraw-Hill; 2004: 194).

EXAM TIP

Hypovolemia and hemoconcentration can lead to an elevated hematocrit and decreased left ventricular end-diastolic volume that result in decreased cardiac output and low-flow state.

EXAM TIP

Burn patients are susceptible to Curling's ulcer, which is due to lack of the normal mucosal barrier.

EXAM TIP

The most common compartment requiring fasciotomy is the anterior tibial compartment.

Fasciotomy

- Escharotomy may fail, especially when the burn is from high-voltage electrical injury or is associated with soft tissue, bone, or vascular injury.
- If compartment syndrome persists after escharotomy, incision of the fascia is also required.
- General anesthesia is required.

Debridement And skin grafting

- Excisional treatment is indicated for most deep second- and third-degree burns once the patient is stabilized.
  - Advantages: Decreased length of stay, earlier return to work, decreased incidence of infection, decreased complications, improved survival.
- Technique:
  - Full-thickness burns require debridement to the investing fascial layer using the scalpel and bovie.
  - Tangential excision may be used for deep partial burns of < 20% BSA and for staged excision of more extensive partial- and full-thickness burns.
  - A Goulian knife, which takes off sequential thin layers, can be used for partial- and some full-thickness excisions; requires debridement until uniform capillary bleeding.
  - Once debridement is complete, the wound is covered with split-thickness skin graft (STSG), full-thickness graft, or biologic dressing.
  - Wound closure.
  - STSG may be applied when the burn is excised or there is no residual nonviable tissue, no pooled secretions, and surface bacterial count is $< 10^5/cm^2$.
  - Autograft should be 0.010–0.015 inches thick:
    - Donor sites may be re harvested (after 2–3 weeks when reepithelialization is complete), but the quality decreases each time as dermis is thinner.
    - Grafts may be meshed at a ratio of 1.5:1 to increase coverage, unless burns are on face or joints.
    - If the risk of mortality is anticipated to be < 50%, first graft hands, feet, face, and joints.
    - If > 50%, graft fat surfaces first to decrease uncovered surface area.
  - Biologic dressings: Bilaminate; outer layer with pores to permit water vapor but not liquid or bacterial passage, and inner layer that permits ingrowth of fibrovascular tissue from wound surface.

OR TIP

Limit burn excision operations to excision of < 20% BSA at one trip to the OR or to a set time limit of 2 hours.

OR TIP

Burn debridement with the Goulian knife is frequently accompanied by a significant amount of blood loss, which can be minimized by using topical thrombin spray and infiltration with epinephrine or vasopressin.

Infection

- Infection of a burn wound causes an increase in the depth (thus converts a second-degree burn to a third-degree).
- Biopsy of the wound is the most definitive way to diagnose burn wound infection.
- Burn patients are very prone to pneumonia and catheter-related infections.

treAtment

- For invasive infection, use topical antimicrobials (see Table 18-1) and start systemic antibiotics.

- For pseudomonal or pediatric infections, infuse subeschar piperacillin, and plan for emergent operative debridement within 12 hours.
- For candidal infections, start antifungal creams; if that treatment fails, start systemic therapy with amphotericin B.

Inhalational Injury and Carbon Monoxide Poisoning

- In closed space burns.
- Carbon monoxide impairs tissue oxygenation by decreasing oxygen-carrying capacity of blood, shifting oxygen-hemoglobin dissociation curve to the left, binding myoglobin and terminal cytochrome oxidase.
- Symptoms do not correlate well with carboxyhemoglobin levels, but levels up to 10% are typically asymptomatic.

signs And symPtoms

Patients may have hoarse voice, cough, wheeze, bronchorrhea, hypoxemia, carbonaceous sputum, head and neck burns, singed nose hairs.

diAgnosis

- Often delayed but is made with the use of chest x-ray (CXR), bronchoscopy, CT scan, and ventilation-perfusion (V/Q) scan.
- Measure carboxyhemoglobin with ABG.
- Bronchoscopy reveals edema and ulceration.
- V/Q scanning will demonstrate carbon particle deposition on endobronchial mucosa.

treAtment

- Half-life of carbon monoxide decreased from 4 hours on room air to 45–60 minutes with 100% oxygen and 15–20 minutes at 3 atmospheric pressure (hyperbaric oxygen).
- Mild injury: Use warm humidified oxygen and an incentive spirometer.
- Moderate: Repeated bronchoscopy when there is continued mucosal sloughing and the patient is unable to clear it.
- Severe, with progressive hypoxemia: Intubation.
  - Nebulized bronchodilators and N-acetylcysteine are useful adjunct treatments.

comPlicAtions

Most commonly, pneumonia.

Electrical Injury

definition

- Extent of injury depends on voltage of current:
  - Up to 1,000 volts—increased resistance limits further passage of current and heating of tissue.

WARD TIP

Infection is more likely in patients with > 30% BSA burn without complete excision or grafting. Apparently infected wounds need to be examined daily and biopsied, including eschar and underlying unburned tissue.

EXAM TIP

Patients with evidence of inhalation injury (soot around nares or mouth) should be intubated.

WARD TIP

Mortality of burns with inhalational injury is increased compared to burns without inhalational injury.

EXAM TIP

Carbon monoxide is a colorless, tasteless, odorless gas and has an af nity for hemoglobin 200 times that of oxygen.

WARD TIP

Normal carboxyhemoglobin: 3–5% nonsmokers; 7–10% smokers.

EXAM TIP

Electrical injuries also result in muscle contractions that can cause injuries like posterior dislocation of shoulder.

EXAM TIP

Extent of damage from electrical injury depends on both current density and tissue conduction. The surface wound usually underestimates the extent of damage to deeper tissues.

WARD TIP

5 Ps of compartment syndrome:

- **P**ain
- **P**aresthesia
- **P**allor
- **P**ulselessness
- **P**oikilothermia

- More than 1,000 volts—passage of current is not limited and tissue injury can continue.
- Deeper tissue may be more severely injured because it cools more slowly.

Mechanism

- Tissue damaged via conversion to thermal energy.
- Damage occurs in skin and underlying tissues along the course of current.
- Skin at the point of contact is often severely charred.

Signs and Symptoms

- Charring at point of contact.
- Myoglobinuria (with muscle damage).
- Hyperkalemia (due to tissue necrosis).
- High-voltage or lightning injury may cause cardiac arrest.
- Neuropathy (immediately following injury, likely to resolve over time).
- Compartment syndrome: Swelling of injured extremity with pain, paresthesia, pallor, pulselessness, poikilothermia.

Treatment

- Patients with loss of consciousness or abnormal ECG require cardiac monitoring for 48 hours even if there are no further arrhythmias or ECG changes.
- Electrical injury is more likely than other types of burn injury to necessitate fasciotomy because deep tissue edema may be extensive, causing compartment syndrome.

Late Complications

- Delayed hemorrhage because of inadequate wound exploration, debridement, or exposure of vessel.
- Cataracts.

Chemical Injury

WARD TIP

Strong acid burns may leave a deceptively healthy appearance of tan smooth skin.

Mechanism

- General: Protein denaturation and precipitation, with release of thermal energy.
- Liquefaction necrosis (alkali).
- Delipidation (petroleum products).
- Vesicle formation (vesicant gas).

Severity

Determined by:

- Concentration
- Amount of agent in contact.
- Duration of contact (tissue injury continues to occur as long as the agent remains in contact with the skin).

Priorities in treAtment

- Remove all clothing to prevent further contact.
- Copious water lavage: Irrigation should continue for at least 30 minutes for acid burns, and longer for alkali burns (because they penetrate deeper into the tissue).
- Check pulmonary status (for edema, mucosal desquamation, bronchospasm).

Nutrition of Burn Patients

- Enteral route preferred (total parenteral nutrition only if enteral is not possible).
- Early feeding recommended.
- High calorie (30–35 kcal/kg/day) and high protein (1.5–2 g/kg/day) requirements.

WARD TIP

Chemical wound irrigation should be with water only. Attempts to neutralize the agent may cause further release of heat, thereby causing further damage.

Burn Scar Cancer

- Rare long-term complication of burn scars (can occur in scars of any origin).
- Called Marjolin's ulcer.
- Usually squamous cell carcinoma, which metastasizes via lymph nodes.
- Diagnosis made by biopsy.
- **Treatment:** Wide excision.

Section iii

High-Yield Facts For surgical electives

Anesthesia	295	Cardiothoracic Surgery	381
Vascular Surgery	301	Transplant	401
Pediatric Surgery	319	The Genitourinary System	413
Ear, Nose, and Throat Surgery	335	Orthopedics	429
Neurosurgery	361	The Hand	445

anesthesia

General Anesthesia 296
Definition 296
Stages of anesthesia 296
asa Classification of Surgical Patients 296
induction 296
neuromuscular Blockade 296
maintenance 298
emergence 298
anatomy 298
Spinal anesthesia 298
epidural anesthesia 298
tap (transversus abdominis Plane) Block 298
local anesthesia 299
Malignant Hyperthermia 299
Definition 299
etiology 300
Signs and Symptoms 300
treatment 300

General Anesthesia

Definition

- Drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation.
- Ability to independently maintain ventilatory function is often impaired; therefore, airway assistance by way of intubation, laryngeal mask airway, or other adjunct is usually necessary.

Stages of anesthesia

Anesthetic drug effects can be divided into four stages of increasing depth of central nervous system (CNS) depression:

Stage I: Analgesia stage
Stage II: Excitement stage
Stage III: Surgical anesthesia stage
Stage IV: Medullary depression stage

ASA Classification of Surgical Patients

See Table 19-1.

Induction

Induction anesthetics can be both IV and inhaled.
See Table 19-2.

Neuromuscular Blockade

- It is important to realize that muscle relaxation does not ensure unconsciousness, amnesia, or analgesia.
- Postsynaptic receptors: Located in the neuromuscular junction, they are the site of action of neuromuscular blockers, blocking acetylcholine (ACh) transmission.

Table 19-1. ASA Physical status classification system and expected Mortality

P1	A normal healthy patient (mortality: 0.1%)
P2	A patient with mild systemic disease (mortality: 0.2%)
P3	A patient with severe systemic disease (mortality: 1.8%)
P4	A patient with severe systemic disease that is a constant threat to life (mortality: 7.8%)
P5	A moribund patient who is not expected to survive without the operation (mortality: 9.4%)
P6	A declared brain-dead patient whose organs are being removed for donor purposes

Reproduced, with permission, from Brunicardi Fc, Andersen DK, Billiar t R, et al. Schwartz's Principles of Surgery. 8th ed. n ew York: McGraw-Hill; 2004: 1860.

WARD TIP

The **8 Ps** of rapid-sequence intubation:
Prepare = equipment
Pretreat = drugs
Position = sniffing position
Preoxygenate = 100% O_2
Pressure = Sellick
Paralyze = drugs
Placement of the tube
Position of the tube = confirm by two methods

WARD TIP

Actually seeing the tube pass between the vocal cords (direct visualization) is the best method to confirm endotracheal tube (ETT) placement. The next best method is end-tidal CO_2.

EXAM TIP

Succinylcholine is contraindicated in the following situations:
- Hyperkalemia
- Burns
- Rhabdomyolysis
- Narrow-angle glaucoma
- Guillain–Barré syndrome
- Malignant hyperthermia
- Other special neuropathies

TABLE 19-2. Anesthetic Agents, Their Actions, and Their Clinical Uses

Effect	Monitor	iv Drugs	Potent gases	Weak gas	Local anesthetics[c]	
Unconscious (amnesia) (anxiolysis)	EEG and/or clinical signs	Benzodiazepines Midazolam Lorazepam Diazepam Barbiturates Etomidate Ketamine[a]	Sevoflurane Desflurane Isoflurane Enflurane Halothane	Nitrous oxide[b]		
Analgesia	Heart rate Blood pressure Respiratory rate	Opioids Fentanyl Morphine Hydromorphone Nonopioid Ketamine[a] Parecoxib Dexmedetomidine Acetaminophen IV Ketorolac	Sevoflurane Desflurane Isoflurane Enflurane Halothane	Nitrous oxide[b]	Amides Lidocaine Bupivacaine Ropivacaine Prilocaine Mepivacaine	Esters Cocaine Procaine Chloroprocaine Tetracaine Benzocaine
					Peripheral nerve blocks (brachial plexus, femoral, etc.) Central nerve blocks (spinal, epidural)	
Muscle relaxation (paralysis)	Nerve stimulator	Depolarizer Succinylcholine Nondepolarizers Pancuronium Vecuronium Rocuronium Atracurium	Sevoflurane Desflurane Isoflurane Enflurane Halothane			

EEG = electroencephalogram; IV = intravenous.

[a]Note that the IV agents are quite specific in their effects, except for ketamine, which has both amnestic and analgesic qualities.

[b]The potent inhalational anesthetics contribute to all three components of anesthesia, but nitrous oxide has weak amnestic and analgesic properties and provides no muscle relaxation at all.

[c]The local anesthetics produce excellent analgesia and muscle relaxation but contribute nothing to amnesia or anxiolysis; these anesthetics must be supplemented with an IV sedative.

Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015. Table 46-1.

- Depolarizing muscle relaxants (succinylcholine) act as ACh receptor agonists, whereas nondepolarizing muscle relaxants (pancuronium, vecuronium, atracurium) function as competitive antagonists.
- Atracurium and cis-atracurium undergo degradation in plasma at physiological pH and temperature by organ-independent Hofmann elimination and so can be used in patients with hepatic and renal dysfunction.
- Anticholinesterases prevent breakdown of ACh, thus allowing ACh to compete more effectively with the paralyzing agents. Can be used to "reverse" neuromuscular blockade of nondepolarizing agents (at end of surgery).

WARD TIP

Nitrous oxide can diffuse into closed spaces/lumens, causing expansion of bowel or worsening of pneumothorax.

WARD TIP

During emergence, patient is at risk for aspiration and for laryngospasm (involuntary spasm of laryngeal musculature, resulting in the inability to ventilate the patient).

WARD TIP

Halothane hepatitis is an immune-mediated hepatotoxicity associated with use of halothane (an inhalational anesthetic). Incidence is 1/22,000 to 1/35,000.

EXAM TIP

Platelet dysfunction is a qualitative defect that affects bleeding time only. Does not affect prothrombin time (PT) or partial thromboplastin time (PTT).

WARD TIP

It is important to limit intraoperative fluid replacement in ESRD to prevent risk of pulmonary congestion.

maintenance

Involves use of inhalational agents (e.g., nitrous oxide, isoflurane, sevoflurane, desflurane) and IV agents (e.g., opiates, ketamine, propofol).

emergence

- Return of patient to an awake state.
- Patient should have full muscle strength and protective airway reflexes before extubation.

Airway: Due to anatomy of pediatric airway, intubation can be more challenging (see Table 19-3).

anatomy

- Subarachnoid space lies between arachnoid and pia mater, which contains cerebrospinal fluid (CSF).
- Spinal cord ends at L1–L2.
- Iliac crests are at level of L4.

Spinal anesthesia

- For surgeries of lower extremities, lower abdomen, genitourinary, and anal region.
- Insert needle at L3–L4 or L4–L5 (at this level cauda equina is present and spinal cord has already ended).

epidural anesthesia

- Indicated for same surgeries as with spinal anesthesia.
- Differs from spinal anesthesia in that needle is placed in epidural space (outside of CSF); commonly an indwelling catheter is left in place.
- With catheter, can have continuous anesthesia (can periodically bolus the catheter for prolonged surgeries).
- Takes longer to obtain effect and requires higher dosages than spinal anesthesia.
- Technically more difficult and requires more time to place than spinal anesthesia.

tap (transversus abdominis plane) block

- Local anesthesia is used for postoperative pain relief in abdominal surgeries. Best done with ultrasound guidance.

table 19-3. Ways Pediatric airway differs from adult

Larger occiput
Large tongue
Larynx is higher up (c3 vs. c4–5) and funnel shaped so narrowest portion of airway is beyond what you see!
Vocal cords are at a slant and more anterior

Table 19-4. Complications of Spinal and Epidural Anesthesia

Complication	Spinal	Epidural
Hypotension (from sympathetic nervous system blockade)	More common	Less common
Nausea (from unopposed parasympathetic activity)	More common	Less common
Postspinal headache (due to CSF leak)	Less common	Occurs only with inadvertent dural puncture during epidural placement
Urinary retention	Common	Common
Backache	Common	Common
Permanent neurologic injury	Very rare	Very rare
Epidural abscess or hematoma	N/A	Rare

Complications

See Table 19-4.

Local Anesthesia

- Used for local infiltration of operative site, during spinal or epidural anesthesia, and peripheral nerve blocks.
- Classification: Ester or amide anesthetic.
- Mechanism of action: Through blockade of sodium channels.
- Myelinated fibers: More susceptible to blockade.
- Sensory fibers: More readily blocked than motor nerves (dose needed for motor blockade is usually double than that needed for sensory block).
- Nonionized form: Needed to cross nerve sheath while the ionized form is the active form.
- Acidosis: Local tissue acidosis as from infection increases ionized drug form and limits anesthetic activity of the drug. Therefore, local infiltration into an area of infection may not produce adequate analgesia.
- Epinephrine: Adding this to the local anesthetic prolongs duration of action by vasoconstriction. Avoid epinephrine in areas with lack of collateral blood flow.
- Toxicity: Initially CNS effects (tinnitus, restlessness, vertigo, seizures), then cardiovascular effects (hypotension, PR prolongation, QRS widening, dysrhythmias).

Malignant Hyperthermia

Definition

Autosomal dominant inherited hypermetabolic syndrome occurring after exposure to an anesthetic agent (very rare but life threatening).

WARD TIP

Remember, the narrowest part of a child's airway is at the glottic opening, beyond what is visualized with a laryngoscope.

EXAM TIP

In infants, the emergency surgical airway of choice is a tracheostomy, not a cricothyroidotomy.

OR TIP

Obstetric patients are at increased risk for aspiration when general anesthesia is undertaken.

OR TIP

Spinal anesthesia is generally used for surgeries lasting no more than 2–3 hours, since the anesthetic is injected only once.

WARD TIP

Think of spinal anesthesia as placing needle closer (deeper) to spinal canal than epidural anesthesia.

EXAM TIP

Amide anesthetics have "i" before "caine" (e.g., prilocaine, lidocaine, bupivacaine).

OR TIP

Do not use epinephrine in these areas:
s PF-10
s crotum
Penis
Fingers
t oes
ears
nose

etiology

Impaired reuptake of calcium by sarcoplasmic reticulum in muscles.

Signs and Symptoms

- Tachycardia, ventricular dysrhythmias
- Hyperthermia
- Hypercarbia, acidosis
- Hypoxemia
- Muscle rigidity

treatment

- Discontinue anesthetics.
- Benzodiazepines (work fastest to control hypermetabolic state).
- Dantrolene sodium (a calcium channel blocker considered more definitive treatment, but onset of action takes about 30 minutes).

HIGH-YIELD FACTS IN

Vascular surgery

Anatomy 302
- Layers of arterial Wall 302
- types of arteries 302
- Venous anatomy 302

Vascular Physical Exam 302
- inspection 302
- auscultation 302
- palpation 302

Diagnostic Tests 303
- Doppler 303
- Duplex 303
- plethysmography 303
- Segmental Blood pressure measurement 303
- arteriography 303
- Digital Subtraction angiography (Dsa) 304
- Spiral computed tomography (ct) 304
- magnetic resonance angiography (mra) 304

Acute Arterial Occlusion 304
- etiology 304
- Signs and Symptoms of acute arterial insufficiency 304
- Differential Diagnosis 304
- Diagnosis 305
- embolism 305
- thrombosis 305

Chronic Ischemia 306
- presentation 306
- risk factors 306
- aortoiliac Disease 306
- infrainguinal Disease 307
- upper extremity Disease 307
- carotid artery Stenosis 308

Amputations 309
- indications 309
- types of amputations 309
- Basic principles 310

Chronic Venous Disease: Obstruction and Incompetence 310
- Basics 310
- presentation 310
- treatment 310

Aneurysm 311
- Definition 311
- Varieties of aneurysm 311
- abdominal aortic aneurysm (aaa) 312
- other aneurysms 314

Diabetic Disease 314
- Location 314
- problems 314
- treatment 314

Venous Thrombotic Disease 314
- anatomy 314
- physiology 315
- Deep Venous thrombosis (DVt) 315
- postphlebitic Syndrome 316
- Superficial Venous thrombosis (sVt) 316

Vascular Trauma 316
- acute compartment Syndrome (acs) 316
- clues to Vascular injury in the trauma patient 317
- a few facts 317

Anatomy

Layers of arterial wall

- **Intima:** One layer of endothelial cells overlying a matrix of collagen and elastin.
- **Media:** Thick layer of smooth muscle cells, collagen, and elastic fibers.
- **Adventitia:** Collagen and elastin, important component to wall strength.

types of arteries

- **Elastic:** Major vessels, including aorta, subclavian, carotid, pulmonary arteries.
- **Muscular:** Branches from elastic arteries: Radial, femoral, coronary, cerebral.
- **Arterioles:** Terminal branches to capillary beds.

Venous anatomy

- Also three layers, but adventitia is most prominent, and intima and media are generally thin.
- Valves prevent reflux.

Vascular Physical Exam

inspection

Signs of vascular insufficiency:

- Hairless, shiny skin.
- Change of skin color—darkening, mottling, reddening, blanching.
- Nail changes.
- Presence of ulcers or gangrene.
- Edema, erythema.

auscultation

- Auscultation for bruits: Carotid, abdomen, common femoral artery (CFA).

palpation

- Temperature: Cool extremities worrisome for poor circulation.
- Edema.

Pulsatile Masses

- Pulsations of normal aorta are palpable in thin people.
- Examine abdomen with both hands around epigastrium and periumbilical area for abdominal aortic aneurysm (AAA).
- Easily palpable pulses in either lower quadrant indicate distal aortic or common iliac aneurysm.

extremity Pulses

- Include brachial, radial, ulnar, femoral, popliteal, dorsalis pedis (DP), posterior tibial.

- Patient should be reclining in supine position, with full exposure of abdomen and legs.
- Legs should be in gentle extension with feet supported.
- To examine pedal pulses, sit at foot of table facing patient.
- Common femoral artery: Found halfway from pubic tubercle to anterior superior iliac spine. (Remember the mnemonic "NAVEL" from lateral to medial: nerves, artery, vein, (empty), lymphatics.)
- Popliteal artery: Place both hands in the middle of the fossa, with fingertips parallel longways (fossa: area between pes anserinus tendon laterally, medial head of gastrocnemius and biceps tendon medially, and biceps and lateral head of gastrocnemius laterally).
- Pedal pulses:
  - DP found between proximal first and second metatarsal (slight dorsiflexion may facilitate).
  - Posterior tibial found posterior to medial malleolus.

WARD TIP

"Scoring" of pulses:
0 = no palpable pulse
1+ = present, but barely palpable
2+ = normal
3+ = normal, strong
4+ = hyperdynamic, abnormally strong

Diagnostic Tests

Doppler

- Relates average flow velocity to frequency shift.

Duplex

- Combines real-time ultrasound (US) with Doppler analysis.

Plethysmography

Measures volume change in organ or body region:

- Pulse volume recording (PVR): Used with Doppler to assess perfusion of distal extremities, assuming change in volume corresponds to change in arterial pressure; useful to predict healing of ulcers and amputations.

Segmental Blood Pressure Measurement

- **Ankle-brachial index (ABI):** Systolic blood pressure (SBP) ankle (via Doppler)/highest SBP in upper extremities.
  - ≥ 1: Normal
  - 0.5–0.7: Claudication, up to moderate arterial disease
  - ≤ 0.5: Severe arterial disease
  - ≤ 0.3: Ischemic rest pain, gangrene
- Misleading results associated with:
  - Diabetic patients, as calcification of vessels makes them less compressible, thereby elevating results falsely.
  - Collateral flow in long-standing insufficiency may artificially improve results.

Arteriography

- Use of contrast dye with fluoroscopy to delineate arteries.
- Risks: Hemorrhage, allergic reaction to dye, thrombosis of puncture site, embolization of clot, renal dysfunction.
- Dose-independent reactions to dye: Asthma, laryngeal edema, spasm, cardiovascular (CV) collapse.

WARD TIP

If you are not sure whether pulse is palpable, count out pulses with second person palpating an easier artery such as the radial artery. If pulsations are simultaneous, you are likely palpating pulse accurately.

WARD TIP

Contrast contains iodine and is renally excreted. Therefore, you should use < 200 cc of dye, and use with caution in patients with renal dysfunction. Ask patients about iodine and shellfish allergies. Hydrate patients prior to and after contrast use.

EXAM TIP

A pseudoaneurysm does not contain all three layers of the arterial wall.

EXAM TIP

Typical scenario: You are asked to see a patient with bleeding from an angiogram puncture site. She has an oozing, pulsatile expanding mass in her groin at the puncture site. Think: Expanding hematoma. Maintain direct pressure for 30 minutes. If bleeding continues, wound exploration may be indicated.

- **Complications:**
  - Neurologic deficits, secondary to emboli.
  - Bleeding: Hematoma, hemorrhage at site.
  - Decreased pulses compared to pre-angiography: If resolves within 1 hour, may be due to spasm; otherwise, consider arterial injury, clot.
- Postprocedure:
  - Maintain patient supine for at least 6 hours.
  - Check puncture site for hematoma or false aneurysm.
  - Follow neurovascular exam, including mental status.
  - Maintain well-hydrated state.

Digital subtraction angiography (DSA)

- Dye is injected into vein or artery, and computerized fluoroscopy subtracts bone and soft tissue so that only the arterial system is visible.

spiral computed tomography (CT)

- Especially useful for AAA.

EXAM TIP

Arterial occlusion progresses to irreversible ischemia in 6–8 hours, depending on collateral circulation.

magnetic resonance angiography (MRA)

- Allows good visualization of patent distal vessels with minimal flow; also useful for evaluation of carotid bifurcation and abdominal aorta.
- Advantage: No contrast used. Gadolinium causes allergic reactions in fewer patients.
- Disadvantage: Cost, limited ability to visualize arterial calcification.

Acute Arterial Occlusion

EXAM TIP

Given Poiseuille's law, in which the formula includes radius to the fourth power, note that radius is the most important determinant of flow.

etiology

- Embolization: From heart or any proximal artery.
- Trauma: Posterior knee dislocation, long-bone fracture, penetrating trauma.
- Iatrogenic (catheter-related).
- Thrombosis: Atherosclerosis, aneurysm.

signs and symptoms of acute arterial insufficiency

EXAM TIP

The **6 Ps** of acute arterial insufficiency:
Pain
Pulselessness
Pallor
Paresthesia
Paralysis
Poikilothermia

- Paresthesia and paralysis are most important signs because nerves are most sensitive to ischemia. Secondarily, there will be loss of muscle function.
- Pain may not exist in diabetics with neuropathy or if rapidly progressive ischemia occurs with anesthesia.
- Earliest sign in lower extremity will be along distribution of peroneal nerve, with hypesthesia, no great toe dorsiflexion, foot drop.
- Progression in muscle damage: Muscles soft, then doughy, then stiff/hard.
- Once skin is mottled and no longer blanches, tissue ischemia is irreversible. Color change indicates extravasation of blood from capillaries into dermis.

Differential Diagnosis

- Nerve root compression
- Deep venous thrombosis (DVT)
- Infection

Diagnosis

- If diagnosis is certain (i.e., cold, newly pulseless, painful extremity), no further workup is required. Patient should be promptly taken to OR.
- Typically, ABI and/or angiography are performed to confirm the site of the lesion. Currently, on-table angiography is an option in some centers.

Embolism

- Must find source of embolism and reason.
- Patient may have chronic atrial fibrillation or history of myocardial infarction (MI) or valvular disease (mitral stenosis, with valvular vegetation or mural thrombus).
- Atheroembolism: From aorta, iliac, or femoral vessels to distal vessels.
- "Blue toe" syndrome.
- Likely to have palpable pedal pulses anyway because at least one proximal vessel is likely still patent.
- Suspect atheroembolism in patients with no history of peripheral vascular disease (PVD), with digital ischemia and palpable pulses.

Treatment

- See below.
- Address primary problem (i.e., atrial fibrillation).

Thrombosis

- Usually underlying stenosis from atherosclerosis.
- May occur in hypercoagulable states, especially with repeated mild trauma.
- Effects of thrombus vary greatly from no symptoms to severe ischemia depending on extent of collateral circulation.
- All patients should have an angiogram unless ischemia is severe and rapidly progressive.
- Cause suggested by history of PVD, popliteal, or aortic aneurysm.
- On exam, patient may have evidence of PVD such as skin changes or lack of distal pulses on contralateral side.

Treatment

- Thrombectomy or grafting (most common).
- Thrombolytic therapy.
- Success rate: 60–80%.
- Mechanism: Activation of plasmin system causes fibrinolysis and clot dissolution.
- Agents:
  - Streptokinase: Bacterial origin (antigenic), binds plasminogen to make plasmin, not often used.
  - Urokinase: From renal parenchyma, directly activates plasminogen, not currently available in the United States.
  - Tissue plasminogen activator (tPA): From vascular endothelium, directly activates plasminogen.
  - Reteplase: Recombinant tPA, catalyzes cleavage of endogenous plasminogen to form plasmin.
- Indications: Acute occlusion of native vessel or graft.
- Contraindications: History of gastrointestinal (GI) or intracerebral lesion, pregnancy, any contraindication to angiography.
- Method: Catheter placed proximally, diagnostic angiogram performed, catheter advanced into clot, thrombolytic agent administered.

EXAM TIP

Typical scenario: You are asked to localize a lesion on a patient. You can palpate a femoral pulse but no popliteal or pedal pulses on the right side. Left-sided pulses are present. Think: Localize lesion to the vessel above site where pulse is first lost. The lesion is likely to be in the superficial femoral artery (SFA). Tissue ischemia will extend one joint level distal to segment of artery occluded.

WARD TIP

Sites commonly affected by embolism:

- Lower extremity: 70% (CFA 34%, popliteal 14%)
- Upper extremity: 13%
- Cerebral circulation: 10%
- Visceral circulation: 5–10%
- Aorta: 9%
- Common iliac artery: 14%

WARD TIP

Any patient with PVD is likely to have cardiac disease as well and will require appropriate preoperative or preprocedure medical stabilization and/or optimization.

WARD TIP

Thrombolytic therapy for acute thrombotic occlusion is not done if severe ischemia is present, as it takes time to dissolve clot.

complications

- Intracerebral bleed, catheter site bleed (both infrequent).
- Patient may ultimately require operation to fix underlying problem.

Chronic Ischemia

presentation

- Claudication.
- Rest pain.
- Gangrene.
- Most commonly affects infrarenal aorta, iliac arteries, SFA at adductor canal.

EXAM TIP

If chronic ischemia occurs at locations other than infrarenal aorta, iliacs, or SFA, the patient is likely to have a comorbid disease, such as diabetes (increased risk at profunda femoris and tibial vessels) or an inflammatory disorder (increased risk at axillary arteries).

risk factors

- Systolic hypertension (HTN)
- Cigarette smoking
- Hyperlipidemia
- Diabetes

aortoiliac Disease

Types

- Localized disease of aorta and common iliac disease (10%).
- Localized disease of external iliac artery (25%).
- Multisegmental disease, including infrainguinal (65%).

Differential Diagnosis

- Nerve root compression (lumbosacral), secondary to disc herniation or spinal stenosis.

Diagnostic Tests

- Femoral-brachial pressure ratio.
- Angiogram.
- Axillobifemoral has better patency rates and is often used for unilateral disease.
- Indicated when patient is extremely at high risk for medical complications following intra-abdominal surgery or if graft from prior operation is infected.

EXAM TIP

Leriche syndrome is indicative of aortoiliac disease and presents with claudication, impotence (decreased flow from the internal iliacs), and decreased (or absent) femoral pulses.

Treatment

- Aortofemoral bypass:
  - Five-year patency 90%, 10-year patency 75%.
  - Better patency rates than aortoiliac bypass.
- Aortoiliac endarterectomy: Acceptable for localized atherosclerotic disease when distal vessel is normal.
- Extra-anatomic bypass:
  - Best option: Femoral-femoral bypass (5-year patency 70%).
  - Other options: Axillofemoral, axillobifemoral—used in high-risk patients for intra-abdominal surgery (elderly, hostile abdomen, abdominal infection).

- Percutaneous transluminal angioplasty (PTA): Indicated for short, nonoccluding lesions with less extensive atherosclerosis.
- Five-year patency rate is 90% with secondary interventions.

complications

- Hemorrhage.
- Thrombosis of reconstruction.
- Distal embolization.
- Ischemic colitis.
- Paraplegia from lumbar vessel ischemia.
- Sexual dysfunction (if operation involved abdominal aorta).

infrainguinal Disease

- Most commonly affects SFA at adductor canal.
- Patients have decreased life expectancy regardless of treatment and outcome.
- Ulcers:
  - Arterial insufficiency: Lateral ankle and foot, pale, no granulation tissue.
  - Venous insufficiency: Medial malleolus, pink, granulation tissue, other stigmata of venous disease.

Diagnostic Tests

- Doppler
- ABI
- PVRs
- Angiogram

Management

- Nonoperative:
  - Smoking cessation, increase exercise tolerance, medication.
  - Pentoxifylline: Decreases viscosity, increases flow; unpredictable relief of symptoms but proven to increase microvascular flow.
  - Cilostazol: Type III phosphodiesterase inhibitor, reduces platelet aggregation and increases vasodilation. Can be more effective than pentoxifylline.
- Operative:
  - Revascularization requires adequate inflow, outflow, and conduit.
  - Bypass (femoral-popliteal): Uses polytetrafluoroethylene (PTFE) or saphenous vein, reversed or in situ—reversed saphenous vein graft (RSVG) best patency.
  - Endarterectomy: Limited to short lesions of SFA at adductor canal or origin of profunda.

WARD TIP

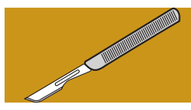
Patients presenting with rest pain, ulcers, or gangrene most likely have multisegmental disease.

WARD TIP

ABI is likely to be artificially elevated in a diabetic patient because the vessels are generally less compressible than normal. PVRs are more useful because results are not affected by compressibility of vessels.

upper extremity Disease

- Compared to lower extremity, more often due to vasospasm or arteritis.
- Palpate pulses: Axillary, radial, ulnar, brachial.
- Subclavian steal syndrome.
- Non hemispheric cerebrovascular symptoms with mild arm claudication due to decreased flow to posterior cerebral artery (PCA) when blood flows retrograde through vertebral artery to SCA.
- If patient has neurologic symptoms, then carotid stenosis is present as well.
- Cause is proximal SCA lesion.
- Angiogram shows flow reversal in vertebral artery.



OR TIP

In situ bypass requires cutting of the valves so that blood flows from proximal to distal.

- Operate for:
  - Incapacitating claudication.
  - Emboli to hand or to posterior cerebral circulation.
  - Symptoms of subclavian steal (if also carotid symptoms, fix carotid first).

Carotid artery stenosis

General

- Differentiate based on asymptomatic or symptomatic.
- Asymptomatic: No history of transient ischemic attack (TIA)-like symptoms.
- Symptomatic history of:
  - Amaurosis fugax—transient blindness due to occlusion of the ophthalmic artery (favorite question of surgeons!).
  - Middle cerebral artery (MCA) syndrome.
  - Anterior cerebral artery (ACA) syndrome.
  - Syncope is not considered "symptomatic" because unilateral carotid occlusion rarely results in impairment of consciousness.

EXAM TIP

Typical scenario: An 83-year-old female with a history of diabetes, HTN, and atherosclerosis presents with painless, monocular vision loss that lasted a few minutes and has now completely resolved. She has no other neurologic deficits. Think: Amaurosis fugax.

Evaluation

Duplex Doppler ultrasound

- Detects increased flow in stenotic lesions.
- Not reliable in very high-grade stenoses.
- Near-complete stenosis results in "trickle" flow.
- May be mistaken for complete occlusion.
- Cannot assess carotids above the mandible.

MRA

- Detects functional flow.
- Can estimate plaque thickness.
- Can evaluate intracranial vasculature.
- May not resolve trickle flow.

EXAM TIP

A carotid bruit detected in isolation is not a reliable indicator of carotid stenosis. In the presence of symptoms suggestive of stroke, a carotid bruit is highly correlated with significant stenosis.

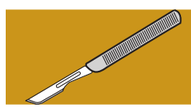
Carotid angiogram

- Gold standard.
- Reveals trickle flow.
- Cannot discern thickness of plaque.

Treatment

- Medical therapy: Aspirin, aspirin plus dipyridamole, clopidogrel.
- Control of underlying medical conditions (HTN, diabetes mellitus, hyperlipidemia).
- Smoking cessation.
- Surgical therapy—carotid endarterectomy (CEA):
  - Indications: Ipsilateral neurologic symptoms with carotid artery stenosis or asymptomatic with > 70% carotid stenosis.
  - CEA has been shown to be more effective than aspirin alone in preventing subsequent strokes in all symptomatic patients. It is superior to medical management alone in asymptomatic females with > 70% stenosis and in asymptomatic males with > 60% stenosis.
  - Emergency CEA may be indicated in symptomatic cases of recent onset with angiographically proven occlusion or loss of a known previous bruit.

- Procedure:
  - Patient is maintained on aspirin preoperatively.
  - Timing is generally 4–6 weeks after CVA.
  - Intraoperative electroencephalogram (EEG) and sensory evoked potentials routinely monitored in case ischemia develops.
  - The surgeon must ensure complete dissection of the plaque in order to prevent remnant plaque rupture with subsequent embolic stroke.
  - After intraoperative ICA occlusion, measurement of retrograde flow from opposite circulation ("stump pressure") should yield mean arterial pressure (MAP) > 50 mmHg. ICA shunt may be indicated if the stump pressure is < 50 mmHg or if the contralateral side also has a tight stenosis.
- Risks/complications:
  - Operative mortality: 1–5%.
  - Vagus is the most common nerve injured (during clamping of the common carotid).
  - Hoarseness—recurrent laryngeal nerve injury.
  - Horner syndrome—injury to sympathetic plexus coursing along with carotids.
  - Partial tongue paresis (hypoglossal nerve injury).
  - Hematoma causing airway compromise.
  - Cerebral hyperperfusion syndrome.
  - Unilateral headache due to poor autoregulation.
  - Seizures.
  - Carotid occlusion (100% stenosis) of chronic nature usually has little benefit from surgical intervention. Typically, slowly progressive carotid stenosis affords development of alternative collateralization to the anterior cerebral circulation (e.g., through external carotid artery [ECA] anastomoses).



OR TIP

Only superficial layers should be closed primarily after CEA. Closure of deep fascia creates an enclosed space capable of retaining hematoma under high pressures. Should an arterial leak develop, the airway may be rapidly compromised.

Amputations

Indications

- Nonviable extremity that has become infected.
- Irreparable vascular injury with irreversible ischemia (traumatic or atraumatic).
- Cancer.
- Elderly patient with infection who is not a candidate for surgical revascularization.



WARD TIP

Carotid artery dissection must always be in your differential in a patient presenting with neurological deficits of the head after a traumatic episode.

Types of Amputations

- **Toe:** For gangrene or osteomyelitis distal to proximal interphalangeal joint (PIP) without proximal cellulitis, necrosis, or edema.
- Transmetatarsal: For necrosis between level of transmetatarsal incision and PIP, often interdigital crease necrosis.
- Syme's: Amputation at base of tibia and fibula. For terminal arterial disease of distal foot—not done very often, requires palpable posterior tibialis pulse.
- Below-knee amputation (BKA) (most common level for nonviable foot): For ischemia up to malleoli.
  - Likely to adapt well to prosthesis.
  - Contraindicated if gangrene more proximal than ankle, or if patient has hip or knee contractures.
  - Likely to fail when there is no femoral pulse.
- Above-knee amputation (AKA): For gangrene above below-knee level, for contractures at knee or hip, and for elderly nonambulatory patients.

- Best chance of healing, particularly in PVD patients.
- Hip disarticulation: For extensive lower extremity ischemia, proximal gangrene, tumor, extensive trauma.
- Poor outcome when performed for PVD.
- Upper extremity amputations: More commonly performed for trauma or tumor.
  - Digital, forearm, upper arm.
  - Leave as much as possible, unless for tumor.

Basic principles

1. Patient should be medically stabilized first when possible.
2. Select level of amputation for PVD:
   - Use PVRs and ABI to help predict chance of healing.
   - At proposed level, infection is controlled, skin looks good, without proximal dependent rubor, proximal pain, and with a venous filling time < 20 seconds.
   - If for cancer, wide excision necessary.
   - If status post trauma or for PVD, remaining tissue level must be acceptably healthy.
   - Consider length of stump in regard to ease of fitting/using prosthesis.
   - Consider overall condition of patient.
3. Determination of closure:
   - Standard: Flaps constructed to close over bone.
   - Guillotine (in situation of sepsis): Level is lower than that eventually desired. Stump left open for dressing changes until sepsis is resolved, at which time completion amputation is performed.
4. Postoperative care:
   - Dressing left in place for at least 3 days unless patient becomes febrile or has profuse bleeding through dressing.
   - Rehabilitation.

WARD TIP

If patient has popliteal pulse, BKA has 90–97% success rate. Without palpable popliteal pulse, success rate is about 82%.

WARD TIP

Longer stump is more functional.

Chronic Venous Disease: Obstruction and Incompetence

Basics

- Postphlebitic syndrome: After DVT, many patients (50%) get chronic venous insufficiency due to valvular incompetence of recanalized veins.
- Up to 50% of patients with signs and symptoms of venous insufficiency have no documented history of DVT.

presentation

- Pain and swelling in affected leg.
- Findings on exam include induration, swelling, skin darkening, and possibly ulcers.

treatment

- Goals:
  - Alleviate symptoms.
  - Heal any ulcers that exist.
  - Prevent development of new ulcers.

WARD TIP

When the patient is compliant and therapy is properly done, 85% of ulcers heal, but may recur.

- Nonoperative:
  - Compression stockings.
  - Leg elevation.
  - Ulcer treatment: Bed rest, antibiotics when cellulitis present, thromboembolism deterrents (TEDs), Unna's boots, dressings.
  - Generalized skin care, with antifungal creams when appropriate, moisturizer for eczema.
- Operative:
- Indications: Severe symptoms, failure of conservative measurements, recurrent ulcers.
- Options:
  - **Ligation** of responsible perforators (usually around medial malleolus).
  - Valvuloplasty (leads to symptomatic improvement in 60–80%).
  - Venous reconstruction with grafting (not popular).



WARD TIP

Unna boots: A wrap for the leg which has been soaked in zinc oxide and calamine lotion. The leg is wrapped from the base of the toes to the tibial tuberosity and left on for a week; it provides compression, healing properties from the zinc, and moisturization. It is used for the treatment of venous stasis ulcers.

Aneurysm

Definition

- An irreversible dilatation of an artery to at least 1.5 times its normal caliber.
- True aneurysm: Involves all layers of arterial wall.
- False aneurysm: Involves only a portion of wall, or involves surrounding tissue.

Varieties of aneurysm

- Degenerative—due to atherosclerosis, the most common type of aneurysm:
  - Intima replaced by fibrin.
  - Media fragmented with decreased elasticity.
  - Imbalance in elastin metabolism, between elastase and α_1-antithrombin.
- Traumatic—due to iatrogenic, catheter-related injury, or to penetrating trauma:
  - Results in focal defect in wall; hemorrhage controlled by surrounding tissue.
  - Formation of fibrous capsule.
- Poststenotic—from Bernoulli's principle, occurs distal to cervical rib in thoracic outlet syndrome, distal to coarctation of aorta, or to aortic or pulmonary valvular stenosis.
- Dissecting—blood travels through an intimal defect, creating a false passageway between the intima and the inner two thirds of the media:
  - If ruptures externally, patient exsanguinates.
  - Risk factors: HTN, Marfan's syndrome, Ehlers–Danlos syndrome, cystic medial necrosis, blunt trauma, cannulation during cardiopulmonary bypass.
- Mycotic—infected.
- #1 agent Salmonella, followed by Staphylococcus.
- Anastomotic—separation between graft and native artery, with formation of sac and fibrous capsule, often in CFA status post aortofemoral bypass:
  - Painless, pulsatile, groin mass.
  - Diagnose with duplex for peripheral aneurysms, and CT for abdominal aneurysms, or angiogram.

abdominal aortic aneurysm (aaa)

- Fusiform dilatation of abdominal aorta more than 1.5 times its normal diameter.
- Normal aorta 2–3 cm.
- Due to degeneration of the medial layer.
- Incidence: 2% of elderly population (nine times more common in males than females).
- Seventy-five percent are asymptomatic at diagnosis.
- If symptomatic, can present with rupture, distal embolization, compression of nearby organs.
- Risk factors for rupture: Diastolic HTN, initially large size at diagnosis, chronic obstructive pulmonary disease (COPD)

screening

- Abdominal US one time for males age 65–75 with history of smoking.

WARD TIP

Size at which AAA is considered for surgical repair: 5.5 cm.

Indications for repair

- Size at which the risk of rupture exceeds the risk of mortality from the operation (generally about 2–5%).
- Increasing size: > 1 cm per year; monitor with CT/US every 6 months.
- Consider factors that increase risk of rupture (diastolic HTN, COPD).
- Symptoms, including pain over AAA site.

Diagnosis

- Exam: Periumbilical palpable pulsatile mass.
- CT: Provides information about character, wall thickness, and location with respect to renal arteries, presence of leak or rupture.
- Magnetic resonance imaging (MRI): Provides more details than CT or US about the lumen, surface anatomy, neck, relationship to renal arteries.
- US: Used more frequently to follow aneurysm size over time than to assess in acute phase (see Figure 20-1).
- Angiogram: Defines vascular anatomy and is especially important in cases of mesenteric ischemia, HTN, renal dysfunction, horseshoe kidney, claudication.

Preoperative assessment

- Includes assessment of carotid disease and cardiac, pulmonary, renal, and hepatic systems.

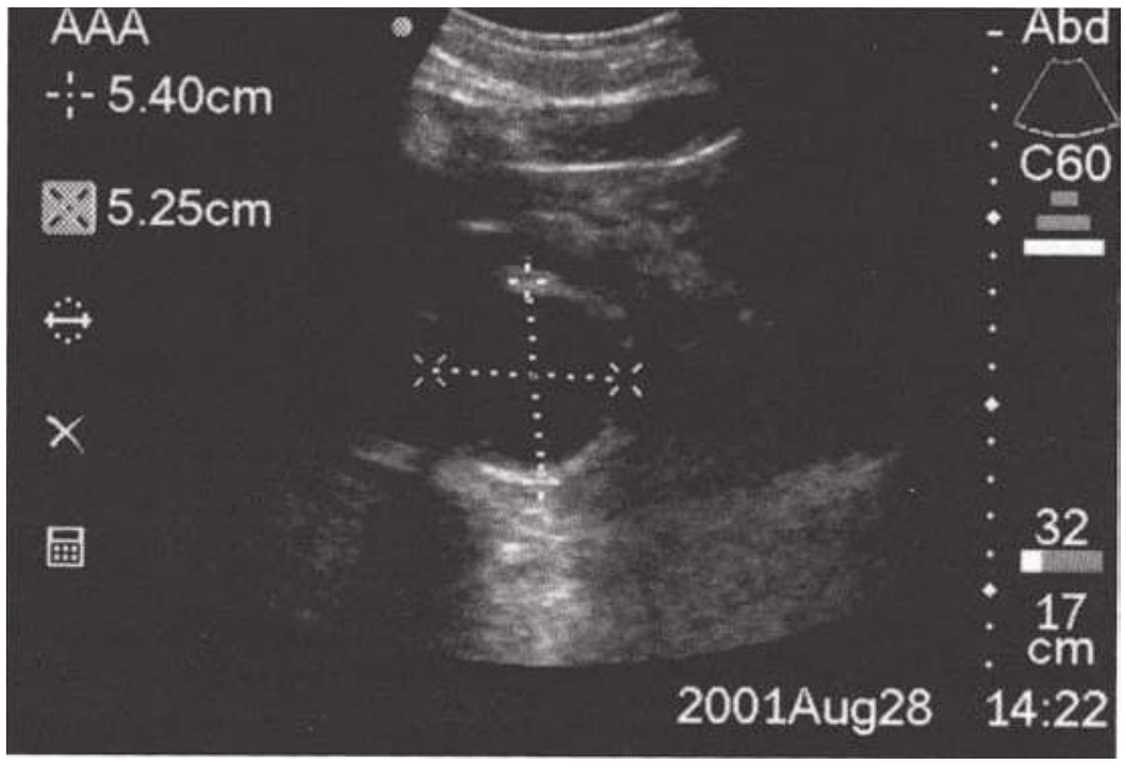


Figure 20-1. aaa on ultrasonography. aortic diameter is 5.40 cm.

- Cardiac workup and optimization:
  - No history of coronary artery disease (CAD): 1–3% chance of MI, pulmonary edema, or cardiac death perioperatively.
  - Known CAD, risks increase to 5–10%.
  - Steps to minimize risk:
    - No cardiac history, normal electrocardiogram (ECG): No further workup.
    - Known but stable CAD: Echocardiogram/dipyridimole or thallium.
    - If abnormal, consider preoperative revascularization.
  - Clinically severe CAD: Definite need for revascularization.
  - If percutaneous, may proceed immediately with AAA repair.
  - If coronary artery bypass graft (CABG), wait 4–6 weeks.

Operative Plan (elective)

- Open repair:
  - Approach: Transperitoneal or retroperitoneal.
    - Retroperitoneal approach: Avoids formation of intra-abdominal adhesions, does not interfere with any GI or genitourinary (GU) stoma that may exist, is better tolerated in COPD patients, and provides better suprarenal exposure.
- Endovascular repair (EVAR):
  - Endovascular system consists of the graft, stents, and a delivery mechanism.
  - Problems include immediate thrombosis and leakage at anatomic sites.
- Should be considered in patients who are elderly and/or have high operative risk due to a multitude of medical comorbidities.
- Benefits compared to open repair: Shorter procedural time, hospital stay, and recovery time; recent evidence suggests improved mortality.

emergent repair of ruptured aaa

- Higher-risk operation.
- **Signs and symptoms:** Abdominal pain, severe back or flank pain, CV collapse.
- **Differential diagnosis:** Perforated peptic ulcer, renal or biliary colic, ruptured intervertebral disk.
- **Treatment:**
  - In an unstable patient with known AAA: Proceed to OR immediately—"mobilize the team."
  - In a stable patient, without history of AAA: CT first, then OR as needed.
  - OR:
    1. Team scrubs and preps/drapes.
    2. Only then is anesthesia induced, as surgeon has knife in hand. (Anesthesia causes vasodilatation, which may worsen hypotension.)
    3. Right angle retractor used to compress supraceliac aorta against vertebrae so patient can be resuscitated by anesthesia.
    4. Retractor replaced with supraceliac clamp.
    5. AAA repaired.

complications of aaa repair

- Renal failure: 6% elective, 75% ruptured—acute tubular necrosis (ruptured) or atheroemboli (elective).
- Ischemic colitis (5%): Bloody diarrhea, elevated white blood count (WBC), peritonitis.
- Spinal cord ischemia: Disruption of artery of Adamkiewicz.
- EVAR: 20–25% will develop endoleak (flow outside graft lumen but inside aneurysm sac).

WARD TIP

Graft is placed within the lumen of the aorta, and the vessels walls are closed around it. The aneurysm is not resected.

EXAM TIP

- Mortality for ruptured AAA is 90%.
- Of those who reach the hospital alive, mortality is still 50%.
- Mortality is further increased in patients with history of CAD, hypotension on arrival, and renal insufficiency, and is also increased by inexperienced surgical team.

EXAM TIP

Typical scenario: You are called to the emergency department to see a 65-year-old male with a history of AAA, for which he is followed by the vascular service. He now complains of abdominal pain and reports syncope at home. Think: Syncope and abdominal pain or hypotension in a patient with known AAA is presumed to be rupture unless proven otherwise. Notify the OR and mobilize your team.

other aneurysms

Iliac artery

- Common iliac artery (CIA), 90%; hypogastric artery, 10%.
- Hypogastric artery aneurysms may cause a pulsatile mass palpable on digital rectal examination.
- Resection indicated when > 3 cm, otherwise 6-month follow-up indicated.
- CIA aneurysm is treated with interposition graft.

EXAM TIP

In pregnant women, iliac artery aneurysms are associated with fibromuscular dysplasia.

Popliteal artery

- Most common peripheral aneurysm.
- Fifty to 75% of cases are bilateral aneurysms.
- Untreated, 60% will lead to thrombosis or distal embolization, and 20% to amputation.
- Diagnosis established on exam, and using US and angiography.
- Operation indicated if no serious comorbidity: Excision and interposition graft with RSVG or ligation and bypass.

EXAM TIP

External iliac artery is never involved when the cause is atherosclerosis.

Femoral artery

- Uncommon.
- On exam, mass, local pain, venous obstruction, embolism, thrombosis.
- Whether symptomatic or not, resection and graft indicated once > 2.5 cm.

EXAM TIP

One third of patients with unilateral popliteal artery aneurysms will have AAAs.

Diabetic Disease

Location

- Usually spares aortoiliac vessels.
- Affects distal profunda femoris, popliteal, tibial, and digital arteries of the foot.
- Small vessels affected: Microangiopathy occurs with intimal and basement membrane thickening.
- Large vessels affected with atherosclerosis and calcification of media.

EXAM TIP

Patients with bilateral popliteal artery aneurysms have a 50% chance of concurrent AAA.

problems

- Diabetic neuropathy: Causes motor and sensory deficits as well as high arch deformity in the foot that increases pressure on tarsal heads (atrophy of intrinsic muscles).
- Arteriopathy.
- Infection: Likely to be worse—gram negatives, gram positives, and anaerobes all potential culprits, in particular Peptococcus, Proteus, Bacteroides.

WARD TIP

ABI in a diabetic patient may be falsely elevated secondary to vessel calcification.

WARD TIP

The diabetic patient may be unaware of skin or other minor injuries. Classic site of infection: Over metatarsal heads on plantar aspect of foot.

treatment

- Revascularization indicated when ulcers occur if there are significant vessels available to use in repair. (See above information for further details that apply to diabetic and nondiabetic patients.)

Venous Thrombotic Disease

anatomy

- Superficial: Greater and lesser saphenous veins.

- Deep: Veins follow arteries and have same names (popliteal, superficial femoral vein [SFV], distal femoral vein [DFV], common femoral vein [CFV], external iliac vein [EIV]).
- Perforators: Connect deep and superficial.

physiology

- Systemic veins contain two thirds of circulating volume.
- Further from heart, there are more valves.
- Venous return depends on respiration: Inspiration causes descent of diaphragm, increased intra-abdominal pressure, and decreased venous return.
- Venous return generated by the relationship among contraction of the heart, static filling pressure, and gravity.
- Virchow's triad: Stasis, blood abnormality, vessel endothelium injury (factors allowing thrombosis to occur).

EXAM TIP

Perforators adjacent to medial malleolus are responsible for stasis ulcers when they become incompetent.

Deep Venous thromBosis (DVt)

- Stasis leads to thrombin formation, which leads to platelet aggregation.
- Endothelial injury and/or a hypercoagulable state may contribute.
- Screening warranted for younger patients or those with repeated occurrences.
- Check prothrombin time (PT), partial thromboplastin time (PTT), WBC, hematocrit (Hct), erythrocyte sedimentation rate (ESR), platelets.
- For extremely high-risk patients, also check homocysteine level, antiphospholipid (APL) antibodies, proteins C and S, antithrombin III (AT III), activated protein C resistance (APC-r), platelet aggregation, mutant factor V Leiden, prothrombin gene mutation (factor II).
- Also check CT scan of the chest, abdomen, and pelvis to look for malignancy if patient has no other risk factors for DVT.

EXAM TIP

The superficial femoral vein is actually a deep vein.

EXAM TIP

About one third of apparently healthy patients with DVTs of unknown cause will be diagnosed with a malignancy within 2 years.

risk Factors

acquireD

- Central venous line placement.
- Congestive heart failure (CHF) and MI (20–40%): Passive congestion and increased blood viscosity.
- Joint replacement operation (15–30%): Femoral vein trauma, long OR, postop immobility.
- Fractures of hip, pelvis, proximal femur (35–60%): Endothelial damage.
- Laparoscopic surgery (7–10%): Intra-abdominal pressure > pressure of venous return from legs.
- Prior DVT (five times): Scarring, valvular damage, decreased muscle pumping, stasis.
- Hormone replacement therapy (HRT) (two to six times): Increases coagulation factors, and decreases protein S, AT III, and fibrinolytic activity.
- Other: Trauma patients, malignancy, obesity, pregnancy, sepsis, prolonged immobility.

congenital

- Antiphospholipid antibody syndrome (APS): Antibodies change normal endothelial function.
- AT III deficiency.
- Plasminogen deficiency: Decreased fibrinolysis.
- Dysfibrinogenemia: Fibrin resistance to plasmin proteolysis, abnormal fibrinogen, defective thrombin binding, increased blood viscosity.



WARD TIP

Central line placement is responsible for one third of upper extremity DVTs.



WARD TIP

Two thirds of DVTs are asymptomatic.

- Factor V Leiden/APC-r: Factor Va resistant to degradation by APC; hyperviscosity syndrome.
- Lupus anticoagulant: Exact mechanism unknown.
- Protein C and S deficiency: Factors V and VIII are not appropriately inactivated.
- Prothrombin gene mutation (factor II).

Diagnosis

- Exam: Calf tenderness, swelling, Homan's sign (not very reliable).
- Duplex: Demonstrates thrombus, assesses compressibility of veins, analyzes venous flow.

Prophylaxis

- TEDs (compression stockings): Increase velocity of venous flow, reduce venous wall distention, and enhance valvular function.
- Sequential compression devices: Reduce incidence of DVT by up to 75% when used properly.
- Pharmacologic: Subcutaneous heparin, low-molecular-weight heparin.

Treatment

- Bed rest until pain and swelling subside.
- Lower extremity elevation.
- Anticoagulation (period of 6 months for first DVT).
- Elastic stockings once ambulating.
- Consider fibrinolytics for large thrombi.

Long-Term Outcome

Recurrent DVT: 18% at 2 years, 30% at 8 years.

Postphlebitic Syndrome

- Occurs in 24%.
- Risk of recurrent DVT then increased six times.
- Symptoms: Edema, pain, aches, fatigue, skin discoloration, scarring, ulcers.

Superficial Venous Thrombosis (SVT)

- Usually a noninfected, localized inflammatory reaction.
- **Signs and symptoms:** Swelling, pain, erythema, tenderness.
- **Diagnosis:** Duplex ultrasonography to rule out associated DVT.
- **Treatment:**
  - Nonsteroidal anti-inflammatory drugs (NSAIDs) as needed until symptoms resolve (about 2–3 weeks).
  - Use elastic stockings.
  - Consider anticoagulation for involvement of saphenofemoral junction.

Vascular Trauma

Acute Compartment Syndrome (ACS)

- Definition: Increased pressure within a compartment with subsequent compromise to the circulation and tissue function.

- Trauma-related etiologies: Fractures (69%), crush injuries, thermal burns, constrictive dressing, injury to vascular structures.
- It is important to remember that non-trauma causes also exist. These involve anything that may increase compartment pressure. (IV fluid extravasation, anticoagulation leading to intramuscular hemorrhage, thrombosis, any ischemic-reperfusion injury.)
- Muscle groups of limbs are divided into compartments by unyielding fascial membranes.
  - Signs and symptoms:
    - The 5 "Ps":
    - Pain out of proportion
    - Pallor
    - Pulselessness
    - Paresthesia
    - Paralysis of muscle associated with that compartment. Ex: ACS of posterior thigh compartment leads to inability to flex the knee (late finding)
- Diagnosis: ACS is a clinical diagnosis. Compartment pressures for ancillary information.
- 0–8 mmHg: Normal pressure
- 25–30 mmHg: Capillary blood flow compromised
- 25–30 mmHg: Onset of pain
- As compartment pressure nears the diastolic pressure, ischemia occurs.
- Diastolic pressure—compartment pressure < 30 mmHg: indication for fasciotomy.
- Management:
  - If high suspicion, proceed with fasciotomy.
  - Prophylactic fasciotomy if concerned:
    - Vascular injury leading to ischemia time of 4–6 hours
    - In concert with some orthopedic procedures (comminuted fractures)
    - Also, remember the basics: Removal of tight dressings, placement of limb at level with heart, analgesics, supplemental oxygen.
  - Complications:
    - Metabolic acidosis from ischemic muscle can lead to myocardial depression, hypotension.
    - Hyperkalemia due to release of potassium from injured muscle.
    - Dessication of open muscle. Avoid with wet-to-dry dressing.

clues to vascular injury in the trauma patient

- Pulsatile or expanding hematoma.
- Pulsatile bleeding.
- Bruit/thrill.
- End-organ ischemia.
- Unexplained shock.
- Likely location.
- Injury to adjacent nerve.
- Extremity fractures with weak or absent pulses.

a few facts

- Carotid artery injury with hematoma can affect cranial nerves IX, X, XI, and XII.
- All zones in neck injury are likely to have vascular injury.
- Limb loss secondary to arterial injury associated with lower extremity fracture > 40%.
- Twenty percent of patients with penetrating abdominal trauma will have major vascular injury.

WARD TIP

Use direct pressure to control bleeding. Apply point pressure using a finger, not the entire hand. Tourniquets are really only a last resort.

Pediatric Surgery

Fluid Management 320

Neck Masses/Lesions 320

- Types 320
- Location 320
- Thyroglossal Duct Cyst and Sinus 320
- Branchial Cleft Cyst and Sinus 320
- Cystic hygroma 321

Congenital Deformities of the Mediastinum and Thorax 321

- Pectus excavatum 321
- Pectus carinatum 321
- Tracheoesophageal (Te) malformations 322
- Congenital Diaphragmatic hernia (CDH) 323
- Pulmonary Sequestration 323
- Congenital cystic adenomatoid malformation (CCAM) 324

Abdominal Wall Defects 324

- gastroschisis 324
- omphalocele 325
- inguinal hernia 326

Gastrointestinal Defects 326

- pyloric Stenosis 326
- Biliary atresia 327
- malrotation and midgut volvulus 328
- intestinal atresia 328
- intussusception 329
- meckel's diverticulum 330
- imperforate anus 331
- hirschsprung's disease (congenital aganglionosis coli) 331
- necrotizing enterocolitis (nec) 332

Neoplastic Disease 333

- Wilms' Tumor 333
- neuroblastoma 334

Fluid Management

Maintenance fluid calculation:

- First 10 kg: 4 cc/kg
- Second 10 kg: 2 cc/kg
- Every additional kilogram past 20 kg: 1 cc/kg
- Minimal urine output: 1–2 mL/kg/hr

WARD TIP

inflammatory masses are the most common congenital neck lesions.
common causes:

- Lymphadenopathy
- Cervical adenitis
- Hematoma
- Benign tumors (lipoma)

WARD TIP

Life-threatening causes of congenital neck lesions:

- Hematoma secondary to trauma.
- Subcutaneous emphysema plus airway injury.
- Non-Hodgkin's lymphoma with mediastinal mass causing airway compromise.

Neck Masses/Lesions

Types

- Congenital
- Inflammatory
- Neoplastic

Location

Location is key in diagnosis.

- **Midline:**
  - Thyroglossal duct remnants
  - Submental lymph node
  - Goiter
- **Lateral:**
  - Branchial cyst
  - Lymphadenitis
  - Lymphoma
  - Carotid body tumor (paraganglioma)
- Midline or lateral:
  - Cystic hygroma
  - Dermoid cyst
- Bilateral:
  - Cervical lymphadenitis

WARD TIP

Thyroglossal duct cysts elevate with swallowing and tongue protrusion, while branchial cleft cysts and dermoid cysts do not move.

Thyroglossal Duct Cysts and Sinus

- See ENT chapter.

WARD TIP

Branchial cleft cysts may not present until adulthood and may become evident only with infection and inflammation.
Probing a branchial cyst can cause infection.

Branchial Cleft Cysts and Sinus

definition

Remnants of the four branchial clefts in the anterior neck that can form cysts or sinus tracts leading through to the skin.

Signs and Symptoms

- Lateral neck mass that may present with drainage.
- Most common form is second branchial cleft cyst that present at the anterior border of sternocleidomastoid muscle.
- Usually painless.
- Fluctuant, mobile, and nontender.

diagnosis

Ultrasound (US).

Treatment

- Complete excision of cyst and entire tract.
- Antibiotics if infected.

Cystic Hygroma

Definition

Congenital lymphangioma.

Signs and Symptoms

- Most are identified at birth.
- May be recognized after injury or upper respiratory infection.
- Painless, soft, and mobile.
- Transilluminate brightly.
- May compress trachea or spread into the floor of mouth, causing upper airway obstruction.
- Do not regress spontaneously.

Diagnosis

Computed tomography (CT) scan (extent and involvement of surrounding structures).

Treatment

Surgical excision is necessary to prevent enlargement and possible airway compromise.

WARD TIP

Cystic hygroma is the most common lymphatic malformation in children and is most commonly found at the floor of the oropharynx.

Congenital Deformities of the Mediastinum and Thorax

Pectus Excavatum

Definition

Abnormal asymmetric development of costal cartilages causing posterior distortion (caving in) of the sternum and xiphoid.

Signs and Symptoms

- Abnormal "funnel chest" appearance.
- May cause respiratory compromise and exertional dyspnea.
- Occasionally associated with mitral valve prolapse.

Treatment

- Surgery at 6 years of age or older.
- Osteotomy to remove abnormal cartilage with a temporary substernal strut to stabilize sternum until new cartilage forms.
- Alternatively: Nuss procedure—only strut placement with no osteotomy.

Pectus Carinatum

Definition

Abnormal asymmetric growth of costal cartilages, causing anterior distortion of sternum and xiphoid; less common than pectus excavatum.

Signs and Symptoms

- Abnormal "pigeon chest" appearance.
- Usually asymptomatic.

treatment

- Brace to correct deformity, can be used up to 18 years of age.
- Surgery (osteotomy) after 6 years of age, usually for cosmetic purposes to avoid psychological distress in children not willing to wear braces.

Tracheoesophageal (TE) malformations

definition

Failure of complete separation of trachea from esophagus. Can occur with or without esophageal atresia and usually involves a TE fistula.

embryology

Esophagus and trachea originate from a single diverticulum and divide at fifth week of gestation.

epidemiology

- One in 3,500 births.
- Most commonly "type C" (esophageal atresia with a distal TE fistula).

Signs and Symptoms

- Respiratory distress or choking following first feeding.
- Excess drooling and salivation.

types (Figure 21-1)

- Pure esophageal atresia.
- Esophageal atresia with proximal TE fistula.
- Esophageal atresia with distal TE fistula.
- Esophageal atresia with proximal and distal fistulas.
- "H-Type"—no esophageal atresia with TE fistula.

diagnosis

- Esophageal atresia becomes evident within the first few hours of life because of severity of symptoms, inability to pass feeding tube.
- Chest x-ray (CXR): Tube to end or coil in the region of thoracic inlet.
- Abdominal x-ray (AXR).
- Contrast x-ray studies.

EXAM TIP

Most common malformation with esophageal atresia is a TE fistula (95%).

EXAM TIP

AXR: Absence of air in stomach. Think: Esophageal atresia without fistula.

WARD TIP

Fifty percent of infants with esophageal atresia have associated anomalies.
Most commonly part of **VACTERL** constellation of anomalies:

- **V**ertebral
- **a**norectal
- **c**ardiac
- **t**racheo**e**sophageal fistula
- **r**enal
- **L**imb

TE malformations also associated with:

- Polyhydramnios
- Preterm birth
- Small for gestational age

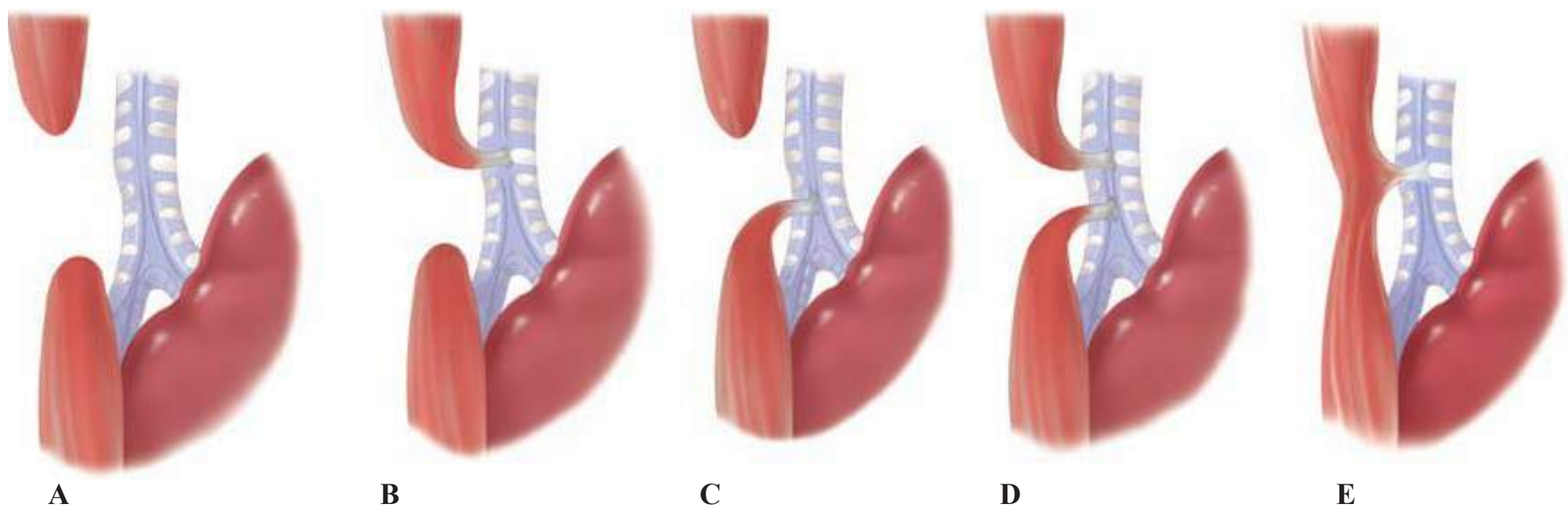


Figure 21-1. Five different forms of te fistula. (Reproduced, with permission, from Brunicardi FC, Andersen DK, Billiar TR, et al. Schwartz's Principles of Surgery. 10th ed. New York, NY: McGraw-Hill Education; 2015: Figure 39-8).

Treatment

- Decompress blind esophageal pouch and control oral secretions with sump tube on constant suction.
- Parenteral prophylactic antibiotics.
- Evaluate for other anomalies (primarily cardiac and renal)—cardiac echo to define aortic arch.
- If cardiac and respiratory stable, may repair surgically.
- Surgical repair:
  - Ligation of fistula and insertion of gastrostomy tube.
  - Anastomosis of two ends of esophagus.

Congenital Diaphragmatic Hernia (CDH)

Definition

Patent pleuroperitoneal canal through the diaphragm leading to pulmonary hyperplasia of the restricted ipsilateral lung and respiratory distress. May result in pulmonary hypertension.

Two types:

- Bochdalek: Posterolateral hernia.
- Morgagni: Anterior retrosternal hernia (in 50% of cases seen with other congenital anomalies).

Signs and Symptoms

- Significant respiratory distress within first few hours of life.
- Scaphoid abdomen.

Diagnosis

- US (prenatally)
- CXR:
  - Bowel gas pattern in hemithorax.
  - Mediastinal shift.

Treatment

- Respiratory and metabolic support (endotracheal [ET] and nasogastric [NG] tubes).
- Gastric decompression.
- Surgical correction by hernia reduction and repair of defect (primary repair or with a synthetic patch).
- If unstable, treated with extracorporeal membrane oxygenation (ECMO) before surgery.

Prognosis

- Survival rate is 50%.
- Predictors of mortality:
  - Pulmonary hypoplasia.
  - Pulmonary hypertension.

Pulmonary Sequestration

Definition

A nonfunctioning embryonic and cystic pulmonary tissue **that receives its own blood supply from the systemic circulation (aorta)** and does not communicate with functional airways.

EXAM TIP

H-type fistula most likely to be seen in emergency department in older children who present with recurrent pneumonias (others usually picked up earlier, while infant is still in hospital).

EXAM TIP

Typical scenario: An infant has excessive oral secretions, choking, and apneic episodes during feedings. Think: TE malformation.

EXAM TIP

Congenital diaphragmatic hernia is more common on the left (as liver blocks the right). Bochdalek is the most common form.

EXAM TIP

Typical scenario: A newborn presents with respiratory distress and a scaphoid abdomen. Think: Diaphragmatic hernia.

WARD TIP

Positive pressure ventilation must be delivered by ET tube, never by mask. Bag and mask ventilation may cause respiratory compromise by inflating gastrointestinal tract.

types

- Intralobar: Contained within normal lung.
  - No pleural covering.
  - Not commonly associated with other anomalies.
  - Present with infection.
  - CXR may reveal mass lesion and air-fluid level.
  - Venous drainage via pulmonary veins.
- Extralobar: Separated from normal lung; can even be under diaphragm.
  - Pleural covering present.
  - Associated with diaphragmatic hernia.
  - Incidental finding on CXR.
  - Venous drainage via systemic veins.

diagnosis

Magnetic resonance imaging (MRI) or aortography to demonstrate sequestration of lung tissue within systemic venous drainage.

treatment

Surgical removal.

congenital cystic adenomatoid malformation (ccam)

definition

Congenital pulmonary malformation involving abnormal cystic lung tissue where normal lung should exist.

Signs and Symptoms

- Neonatal respiratory distress
- Recurrent respiratory infection

diagnosis

CXR:

- Characteristic "Swiss cheese" appearance of affected lung.
- May be difficult to distinguish from air-filled loops of bowel associated with CDH.
- Location of NG tube is key.

treatment

Surgical resection.

Abdominal Wall Defects

gastroschisis

definition

Centrally located, full-thickness abdominal wall defect, leading to exposed bowel.

epidemiology

One in 10,000 live births.

OR TIP

Prior to resection of lobe, it is necessary to examine blood supply to ascertain that it is not anomalously originating from the abdomen; cutting the vessel in this case can lead to its retraction into the abdomen and internal hemorrhaging.

WARD TIP

Congenital cystic adenomatoid malformation is the second most common congenital lung lesion (after lobar emphysema).

EXAM TIP

Presentation of congenital cystic adenomatoid malformation similar to diaphragmatic hernia.

EXAM TIP

Where is the NG tube tip?
Tip in thorax: CDH
Tip in abdomen: CCAM
This makes the difference between a thoracic and an abdominal surgery!

diagnosis

- Often detected during prenatal US, associated with high alpha-fetoprotein (AFP).
- Defect is usually to the right of umbilicus.
- Not usually associated with other anomalies.

treatment

- Temperature regulation.
- Sterile covering with plastic wrap (Silo) to minimize evaporative loss.
- NG decompression.
- Broad-spectrum antibiotics.
- Total parenteral nutrition (TPN).
- Surgical correction and closure of abdomen.

Prognosis

- Greater than 90% survival rate, postsurgical.
- Twenty percent of cases are complicated by necrotizing enterocolitis (NEC).

omphaLoceLe

definition

- Herniation of abdominal contents (often including the liver) into the base of the umbilical cord.
- Protective membrane present.
- Elements of the umbilical cord course individually over the sac and come together at its apex to form a normal-appearing umbilical cord.

epidemiology

- One in 5,000 live births.
- More common in babies born to mothers < 20 years old and > 40 years old.

associated anomalies

- Beckwith–Wiedemann syndrome (gigantism, macroglossia, umbilical defect, hypoglycemia).
- Trisomy 13 and 18.
- Pentalogy of Cantrell (omphalocele, diaphragmatic hernia, cleft sternum, absent pericardium, intracardiac defects).
- Exstrophy of the bladder or cloaca.

types

- Small: Contains only intestine.
- Large: Contains liver, spleen, and gastrointestinal (GI) tract.

treatment

- Ruptured sac:
  - Similar to gastroschisis treatment.
  - Emergent surgical correction.
- Intact sac:
  - Less urgent.
  - Timing of surgery depends on size of defect, size of infant, and presence of other anomalies.

WARD TIP

No difference has been shown in outcomes for gastroschisis or omphalocele whether infant is delivered vaginally or via C-section.

WARD TIP

Seven percent have coexistent intestinal atresia—important to search for this at surgery.

WARD TIP

Ruptured omphalocele may be confused with gastroschisis, but the former does not have an intact umbilical cord at the level of abdominal wall.

Prognosis

Dependent on presence of other anomalies.

WARD TIP

If a Meckel's diverticulum is found within a hernia sac, it is called a Littre's hernia.

inguinal hernia

definition

Protrusion of a viscus or part of a viscus through an abnormal weakening in the abdominal wall and through the inguinal canal. In children, most commonly an indirect inguinal hernia from a processus vaginalis that has remained patent.

epidemiology

- One to 5% of newborns.
- Male-to-female ratio: 4:1.
- Higher incidence in premature infants (30%).
- Indirect hernias are more common on the right side because of the right testicle descends later.

WARD TIP

Correction of inguinal hernia is the most frequent surgical intervention in children.

Signs and Symptoms

Bulge in the groin, scrotum, or labia, especially with increased intra-abdominal pressure (coughing, crying, straining, and blowing up a balloon).

OR TIP

In females and males < 1 year old, the risk of bilateral hernias is greater and the contralateral side should be explored. The other side can be visualized by a laparoscope placed through the hernia sac (transinguinally).

diagnosis

- Physical exam shows firm mass that can slip through the internal inguinal ring.
- Important to differentiate from a hydrocele, which does not extend to the internal ring.
- "Silk glove sign": After hernia is reduced, sac and thickened spermatic cord can be palpated and rolled underneath the examiner's finger.

WARD TIP

Indirect inguinal hernias are located lateral to the inferior epigastric vessels (and direct are medial).

treatment

- Operative repair:
  - Should be repaired shortly after diagnosis except in premature infants.
  - Unlike adults, pediatric cases are usually not repaired with a mesh. In pediatrics, high ligation of the hernial sac (herniotomy) is performed without repair of the abdominal wall.
- Reasons to operate:
  - Major risks include incarceration of a loop of bowel, an ovary, or a fallopian tube.
  - Fifteen to 30% of hernias are incarcerated.
  - Increased risk of incarceration in first few months of life.

Gastrointestinal Defects

pyloric stenosis

definition

Narrowing of the pyloric canal due to hypertrophy of the musculature.

epidemiology

- One in 250 births.
- Male-to-female ratio: 4:1.

causes

Unknown.

Signs and Symptoms

- Usually evident between 2 weeks and 2 months old.
- Nonbilious, projectile vomiting.
- Hungry after vomiting.
- Dehydration.
- Midepigastric mass ("olive").
- Visible peristaltic wave (left to right).
- Hypochloremic metabolic alkalosis with paradoxic aciduria.

diagnosis

- Ultrasound (90% sensitivity):
  - Elongated pyloric channel (> 14 mm).
  - Thickened pyloric wall (> 4 mm).
- Radiographic contrast series:
  - **String sign**—from elongated pyloric channel.
  - Shoulder sign—bulge of pyloric muscle into the antrum.
  - Double track sign—parallel streaks of barium in the narrow channel.

treatment

- Correction of fluid and electrolyte and acid–base balance.
- IV fluid 5% dextrose in normal saline plus potassium chloride 3–5 mEq/kg.
- Surgical correction: Ramstedt pyloromyotomy—dividing the circular fibers of the pylorus without entering the gastric lumen.

EXAM TIP

Hypokalemic, hypochloremic metabolic alkalosis with paradoxical aciduria is seen in pyloric stenosis.

EXAM TIP

Pyloric stenosis:

- Children of affected parents have a 7% chance of disease.
- Most common in firstborn male.

BILIARY ATRESIA

definition

Obliteration of the entire extrahepatic biliary tree at or above the porta hepatis.

Signs and Symptoms

- Neonatal jaundice (beyond first week).
- Hyperbilirubinemia.
- High γ-glutamyl transpeptidase (GGT) level.
- Signs of portal hypertension (hepatosplenomegaly, ascites).

diagnosis

- Radioisotope scanning.
- Ultrasound.
- Direct bilirubin:
  - Greater than 2 mg/dL.
  - Greater than 10% of total bilirubin.

treatment

- Laparotomy, liver biopsy, and operative cholangiography should be done in any suspicious case.
- Correctable type:
  - Blind-ending cystic dilation of the common hepatic duct.
  - Repaired by direct anastomosis with Roux-en-Y loop of jejunum.

WARD TIP

Optimal time of surgery for biliary atresia is < 8 weeks of age.

WARD TIP

Biliary atresia accounts for 90% of extrahepatic obstruction in neonates. Of all causes of neonatal jaundice, biliary atresia is the most common indication for surgery.

EXAM TIP

Biliary atresia is associated with other congenital anomalies in one fourth of cases: duodenal atresia, malrotation, annular pancreas, congenital heart defects.

- Noncorrectable type: Kasai procedure—hepatoportoenterostomy (anastomosis between the porta hepatis and the small intestines to drain bile from liver).
- Postoperative treatment:
  - Prophylactic antibiotics.
  - Phenobarbital.
  - Liver transplantation.

Malrotation and Midgut Volvulus

Definition

- Incomplete rotation of the intestine during fetal development.
- May cause complete or partial duodenal obstruction.

Embryology

- Midgut = duodenum to mid-transverse colon.
- Develops extraperitoneally and migrates intraperitoneally at 12 weeks.
- During this migration, the midgut rotates 270° counterclockwise around the superior mesenteric artery (SMA).
- Problem results from abnormal fixation of the mesentery of the bowel.

WARD TIP

Acute onset of bilious vomiting in an infant is malrotation and volvulus until proven otherwise!

Signs and Symptoms

- Acute onset of bilious vomiting.
- Abdominal distention.
- Lethargy.
- Skin mottling.
- Hypovolemia.
- Bloody stool (late sign).

Diagnosis

- AXR:
  - Presence of bowel loops overriding liver.
  - Air in stomach and in duodenum (double bubble sign).
  - No gas in GI tract distal to volvulus.
- Upper GI series: Duodenal C-loop does not extend to the left (stops at level of duodenum).
- Barium enema: Cecum located in right upper quadrant (RUQ).

Treatment

- Surgical emergency (can lead to intestinal infarction and death if surgery is delayed).
- Ladd procedure:
  - Reduced with counterclockwise rotation.
  - Ligation of Ladd's bands (abnormal fibrous bands attached to cecum and causing obstruction).
  - Appendectomy—because cecum remains in RUQ, changing position of appendix and making appendicitis harder to diagnose.

WARD TIP

Malrotation without volvulus may present with intermittent vomiting and abdominal distention.

Prognosis

Ten percent chance of recurrent volvulus.

EXAM TIP

Duodenal atresia is the most common type of intestinal atresia. Most common site of atresia is at the ampulla of Vater.

Intestinal Atresia

Definition

Failure of the duodenum to recanalize during early fetal life.

associated conditions

- Down syndrome.
- Esophageal atresia.
- Imperforate anus.
- Small for gestational age.
- Polyhydramnios.

Signs and Symptoms

- Bilious vomiting.
- Abdominal distention.

diagnosis

Plain abdominal film:

- Dilated bowel proximal to obstruction.
- "Double bubble" sign (air in stomach and duodenum).

treatment

- Fluid resuscitation.
- Gastric decompression.
- Broad-spectrum antibiotics.
- Duodenal atresia: Side-to-side anastomosis (avoids injury to bile and pancreatic duct).
- Jejunoileal atresia: End-to-end anastomosis.

intussusception

definition

Invagination of one portion of the bowel into itself—proximal portion usually drawn into distal portion by peristalsis.

epidemiology

- Incidence 1–4 in 1,000 live births.
- Male-to-female ratio: 2:1–4:1.
- Peak incidence 5–12 months.
- Age range: 2 months–5 years.

causes

- Idiopathic.
- "Lead point" (or focus) caused by:
  - Hypertrophied Peyer's patches from viral infection (enterovirus in summer, rotavirus in winter).
- In older children:
  - Meckel's diverticulum.
  - Polyp.
  - Lymphoma.
  - Henoch–Schönlein purpura.
  - Cystic fibrosis.

Signs and Symptoms

- Classic triad:
  - Intermittent colicky abdominal pain.
  - Bilious vomiting.
  - Currant jelly stool.
  - Dance's sign—absence of bowel in right lower quadrant (RLQ) with a diffuse sausage shaped RUQ mass.

WARD TIP

Passage of meconium does not rule out intestinal atresia.

WARD TIP

Differential diagnosis includes volvulus and annular pancreas.

WARD TIP

Since malrotation/volvulus has the same radiographic double bubble sign, get upper GI for confirmation.

EXAM TIP

Most common cause of acute intestinal obstruction under 2 years of age.
Most common site is ileocolic (90%).

WARD TIP

Intussusception and link with rotavirus vaccine led to withdrawal of vaccine from the market.

EXAM TIP

intussusception

- Classic triad is present in only 20% of cases.
- Absence of currant jelly stool does not exclude the diagnosis.
- Neurologic signs may delay the diagnosis.

diagnosis

- AXR:
  - Paucity of bowel gas.
  - Loss of visualization of the tip of liver.
  - "Target sign"—two concentric circles of fat density.
- US:
  - "Target" or "donut" sign—single hypoechoic ring with hyperechoic center.
  - "Pseudo-kidney" sign—superimposed hypoechoic (edematous walls of bowel) and hyperechoic (areas of compressed mucosa) layers.
  - Barium enema.

treatment

- Correct dehydration.
- NG tube for decompression.
- Hydrostatic reduction.
- Barium enema:
  - Cervix-like mass.
  - Coiled spring appearance on the evacuation film.
- Contraindications:
  - Peritonitis.
  - Perforation.
  - Profound shock.
- Air enema:
  - Decreased radiation.
  - Fewer complications

recurrence

- With radiologic reduction: 7–10%.
- With surgical reduction: 2–5%.

WARD TIP

Barium enema for intussusception is both diagnostic and therapeutic. Rule of **3s:**

- Barium column should not exceed a height of **3** feet.
- No more than **3** attempts.
- Only **3** minutes/attempt.

meckel's diverticulum

definition

Persistence of the omphalomesenteric (vitelline) duct (should disappear by seventh week of gestation):

- Arises from the antimesenteric border of ileum.
- Contains heterotopic epithelium (gastric, colonic, or pancreatic).
- A true diverticulum in that it contains all layers of bowel wall.

EXAM TIP

Meckel's diverticulum is the most frequent congenital GI abnormality.

epidemiology

- Two percent incidence.
- More common in males.
- Usually presents in children < 2 years old.

EXAM TIP

Meckel's diverticulum:
Occurs in **2**% of population.
Is **2** inches long.
Is **2** feet from the ileocecal valve.
Usually occurs in patients under **2** years of age.
Is symptomatic in **2**% of patients.
Contains one of **2** types of tissue: gastric or pancreatic.

Signs and Symptoms

- Intermittent painless rectal bleeding.
- Intestinal obstruction.
- Diverticulitis.

diagnosis

Meckel's scan (scintigraphy) has 85% sensitivity and 95% specificity. Uptake can be enhanced with cimetidine, glucagon, or gastrin.

Treatment

Surgical: Diverticular resection. If base is thickened then segmental resection of small bowel adjacent to the diverticulum.

WARD TIP

Meckel's diverticulum may mimic acute appendicitis and also act as lead point for intussusception.

Imperforate Anus

Definition

Lack of an anal opening of proper location or size.

Causes

Results from a failure of the urinary and hindgut systems to separate.

Associated Anomalies

VACTERL anomalies (**V**ertebral, **A**norectal, **C**ardiac, **T**racheo**E**sophageal malformations, **R**enal, **L**imb).

Types

High and low: Classification depends on whether the rectum ends above (high) or below (low) the puborectalis sling.

Treatment

- Colostomy for high lesions.
- Perineal anoplasty or dilatation of fistula for low lesions.

Prognosis

The higher the lesion, the poorer the prognosis.

Hirschsprung's Disease (Congenital Aganglionosis Coli)

EXAM TIP

Hirschsprung's disease is the most common cause of lower intestinal obstruction in the neonate.

Definition

Congenital absence of ganglion cells in the Auerbach (myenteric) and Meissner (submucosal) plexi that results in intestinal obstruction.

Epidemiology

- Occurs in 1 in 5,000–8,000 live births.
- Male-to-female ratio: 4:1.
- Family history.

Types

- Rectal.
- Rectosigmoid.
- Entire colon.

Signs and Symptoms

In neonatal period:

- Ninety-five percent present as delayed passage of meconium (> 24 hours).
- Rectal examination.
- An empty vault that is not dilated.
- Explosive release of feces.
- Most ominous presentation is enterocolitis (most common cause of mortality in Hirschsprung's).

- Presentation later in childhood:
  - Bilious vomiting.
  - Chronic constipation.
  - Abdominal distention.
  - Failure to thrive.

diagnosis

- AXR to look for evidence of obstruction.
- Barium enema to look for transition zone (may not be present until 1–2 weeks of age).
- Rectal biopsy for definitive diagnosis (must include submucosa; samples are taken 1, 2, and 3 cm from dentate line, shows absence of ganglion cells).

treatment

Surgical repair:

- Temporary colostomy proximal to transition zone at diagnosis.
- Definitive repair when the infant is 6–12 months old.
- Closure of colostomy 1–3 months postoperatively.

necrotizing enterocolitis (nec)

definition

Serious intestinal injury and necrosis following a combination of vascular, mucosal, and toxic insults to a relatively immature gut.

epidemiology

- Occurs in 1–3 in 1,000 live births.
- Predominantly a disorder of preterm infants; incidence increases with decreasing gestational age.
- Increased incidence with congenital heart disease, severe intrauterine growth retardation (IUGR), sepsis, gastroschisis, and other neonatal disorders.

EXAM TIP

typical scenario: A premature infant born at 33 weeks' gestation now at 1 week of age has developed feeding intolerance, is febrile, and has hematochezia and a distended belly. Think: Necrotizing enterocolitis.

Signs and Symptoms

Presentation is similar to sepsis.

- Systemic:
  - Lethargy.
  - Feeding intolerance.
  - Fever.
  - Hypothermia.
  - Hypotension.
  - Apneic spells.
- GI:
  - Abdominal distention, tenderness.
  - Vomiting.
  - Bloody diarrhea.
  - Hematochezia.

WARD TIP

NEC is the most common GI emergency for infants.

diagnosis

- AXR: Pneumatosis intestinalis (gas within the bowel wall).
- Lateral decubitus or cross-table lateral:
  - Probable NEC:
    - Thickened bowel loop.
    - Fixed position loops on serial films.
    - Ascites.

- Definite NEC:
  - Intramural gas.
  - Portal gas.
  - Pneumoperitoneum/free air (denotes perforation).

Treatment

- Nothing by mouth.
- Bowel decompression.
- Antibiotics/sepsis evaluation.
- TPN.
- Monitor vital signs and abdominal girth.
- Monitor fluid intake and output.
- Definite indication for surgical resection:
  - Perforation.
  - Full-thickness necrosis of bowel.
- Possible indications:
  - Ascites.
  - Deterioration with medical management.
  - Intestinal obstruction.

EXAM TIP

Pneumoperitoneum (free air) on an AXR of an infant with suspected NEC is an indication for immediate surgery!

Prognosis

Variable (depends on extent of injury). Overall > 95% survival rate.

Neoplastic Disease

Wilms' Tumor

definition

Nephroblastoma—originates intrarenally.

epidemiology

- Most common intra-abdominal malignancy in childhood.
- Usual presentation between 2 and 4 years; very rapidly growing.

associated conditions

- WAGR syndrome (**W**ilms' tumor, **A**niridia, **G**enitourinary anomalies, mental **R**etardation).
- Beckwith–Weidemann syndrome (hemihypertrophy, macroglossia, organomegaly).
- Denys–Drash syndrome (Wilms' tumor, pseudohermaphroditism, and glomerulopathy).

WARD TIP

Presence of abdominal mass in infant or child is always abnormal and requires evaluation.

EXAM TIP

Wilms'tumor is associated with aniridia, Beckwith–Wiedemann syndrome, hemihypertrophy, neurofibromatosis, horseshoe kidney.

EXAM TIP

Beckwith–Wiedemann syndrome consists of omphalocele, macrosomia, organomegaly, macroglossia, Wilms' tumor, and neonatal hypoglycemia.

Signs and Symptoms

Triad:

- Flank mass.
- Hematuria.
- Hypertension.

diagnosis

- CT scan with contrast (metastasis—nodal enlargement and liver nodule).
- CXR or CT scan (lung metastasis).
- Intravenous urography (intrarenal solid mass).
- Ultrasound of abdomen (extension into renal vein and inferior vena cava [IVC]).

WARD TIP

Lung is the most common site of metastasis.

Stages

I: Involves only kidney.
II: Invades capsule and possibly perirenal fat.
III: No hematogenous metastasis, not completely resectable.
IV: Hematogenous metastasis to lung, brain, distal nodes.
V: Involves both kidneys.

treatment

- Surgical resection of tumor, exploration of abdomen, evaluation of contralateral kidney.
- Chemotherapy + radiotherapy (for later stages).
- Ninety percent survival rate.

neuroBLastoma

definition

- Arises from neural crest cells.
- May arise in adrenal medulla, sympathetic ganglia, and organ of Zuckerman (para-aortic ganglia).

WARD TIP

Most common tumor to metastasize to the orbit, causing apoptosis in the affected eye.

epidemiology

- Most common solid tumor in infants (> 33%: < 1 year old, 80%: < 5 years old).
- Most common solid tumor of childhood outside of central nervous system.
- Abdominal tumors are most common presentation: 65% in adrenal gland.
- Thoracic tumors are next most common presentation.

WARD TIP

More than 50% have metastases when first seen.

Signs and Symptoms

- Abdominal pain and mass.
- Fever, failure to thrive, diarrhea, and lethargy.
- Neurological symptoms (ataxia, opsomyoclonus).
- Hypertension (25%).

diagnosis

- Urine: Raised catecholamines (vanillylmandelic acid [VMA], homovanillic acid [HVA]).
- Intravenous pyelography (inferior displacement of opacified calyces—"drooping lily sign").
- CT abdomen with contrast.

WARD TIP

Neuroblastoma can cross the midline, while Wilms' tumor does not.

Stages

I: Tumor is limited to one organ, completely resectable.
II: Tumor does not cross midline.
III: Crosses midline.
IV: Distant metastases.

WARD TIP

Better prognosis for neuroblastomas with younger age (< 1 year old), fewer N-myc proto-oncogene copies.

treatment

- Stage I: Resection.
- Stages II and III: Resection and chemotherapy.
- Stage IV: Resection and aggressive chemo/radiotherapy.

HIGH-YIELD FACTS IN

Ear, Nose, and Throat Surgery

Ear 336
Anatomy 336
Physiology 337
Pathology 338

The Facial Nerve 340
Facial nerve Paralysis 340

Nose 341
Anatomy 341
Physiology 342
Sinus Disease 342
epistaxis 343
nasal masses 343

Oral Cavity and Oropharynx 344
Anatomy 344
Physiology 345
ranula 346
oral malignancies 346

Larynx and Hypopharynx 347
Anatomy 347
Physiology 348
Benign lesions of the vocal cords 349
laryngeal cancer 350

Salivary Glands 351
Anatomy 351
Physiology 351
Benign/systemic disease 352
Benign tumors 353
malignant tumors 354

Neck 354
Anatomy 354
congenital diseases in the neck 357
infectious/inflammatory lesions of the neck 358
malignancy 359

References 360

Ear

Anatomy

External Ear

See Figure 22-1.

- Auricle: Funnels sound waves into external auditory canal (EAC).
- External auditory canal: The outer one third is cartilaginous; the inner two thirds is osseous.
- Tympanic membrane (TM) is divided into two parts: Pars flaccida superiorly and pars tensa inferiorly. TM is attached to the malleus medially, and conducts sound waves from external ear to middle ear.

Middle Ear

Contains the eustachian tube orifice, ossicles, and entrance to the mastoid cavity:

- The eustachian tube connects the nasopharynx to the anterior wall of the middle ear cavity. It serves to equalize middle ear pressure to atmospheric pressure. The adenoids are in close proximity to nasopharyngeal opening of tube.
- Ossicles: Tympanic membrane → malleus → incus → stapes → oval window → perilymph of scala vestibuli of the inner ear. The ossicles serve as a lever arm to amplify sound. The malleus is fused to the TM internally, and with the incus medially. Tensor tympani muscle is attached to the manubrium of the malleus. The **incus** articulates with the other two ossicles. No muscular attachments. The neck of the **stapes** serves as the attachment for the stapedius muscle, innervated by the stapedial branch of cranial nerve (CN) VII. The footplate of the stapes is attached to the oval window.

EXAM TIP

The tensor tympani makes tympanic membrane taut and amplifies sounds; the stapedius pulls the head of the stapes back and decreases sound intensity.

Inner Ear

The inner ear labyrinth consists of a bony labyrinth encompassing the membranous labyrinth and organs of hearing and balance.

- **Acoustic apparatus:** Composed of the cochlea, which consists of the scala vestibuli and scala tympani, both of which are filled with perilymph. The

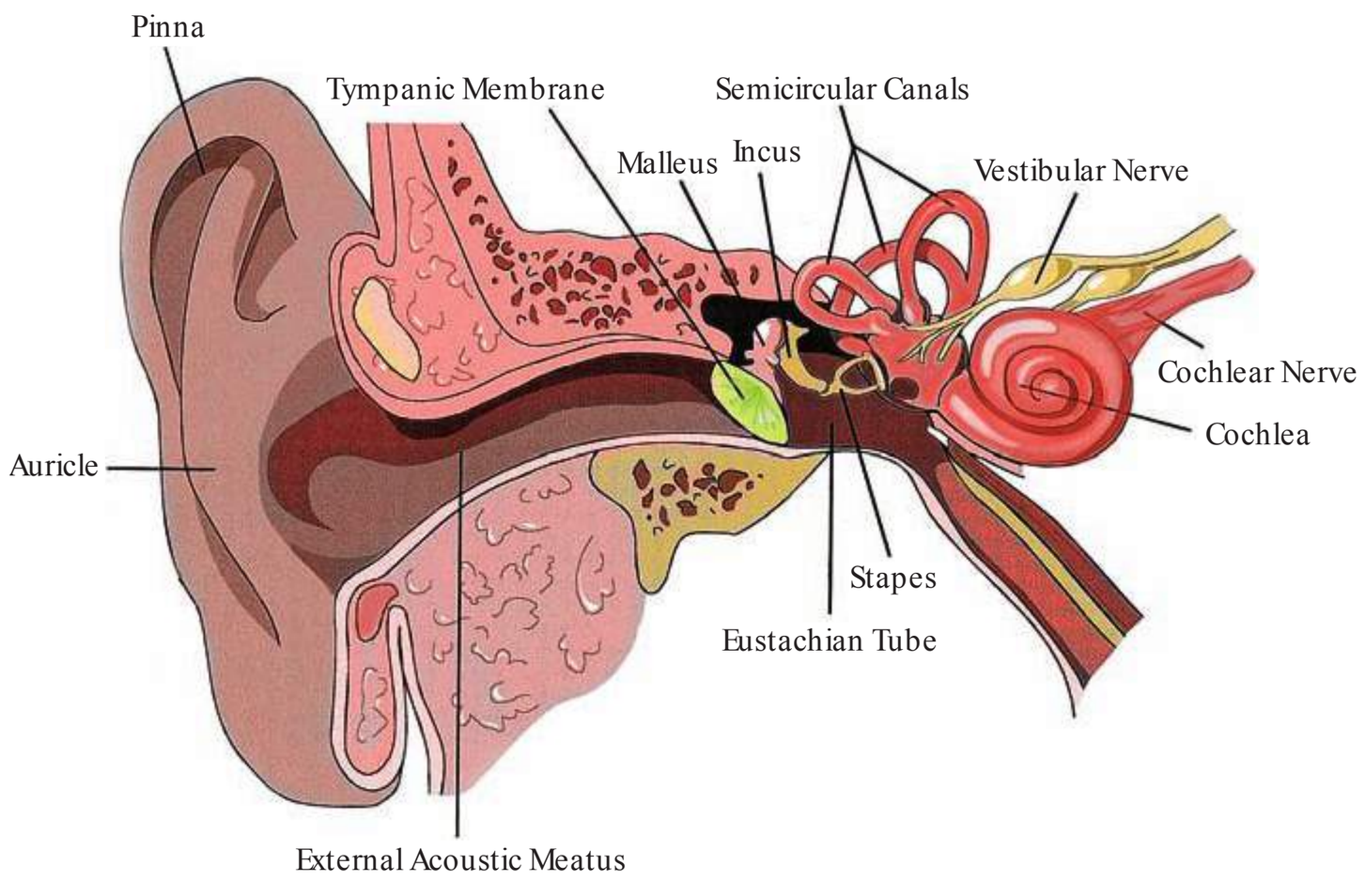


FIgurE 22-1. anatomy of the ear.

oval window transmits vibrations to the scala vestibuli at the base of the cochlea, which, via the helicotrema at the apex of the cochlea, transmits the pressure waves to the scala tympani. The scala tympani transmits the waves to the round window, where they are dissipated. The scala media, which contains the spiral organ of Corti, is the sensory portion of the inner ear and is filled with endolymph. The inner and outer hair cells in the organ of Corti transmit signals to the cochlear nerve (CN VIII). The neuroepithelium in the cochlea is arranged in a ribbon-like structure along the entire length of the cochlea, with tonotopic organization of sounds—higher frequencies are represented at the base of the cochlea, lower frequencies at the apex.

- **Vestibular apparatus:** Includes the vestibule (utricle and saccule) and semicircular canals, which provide input concerning gravitational pull/linear acceleration and angular acceleration, respectively. Cells in the neuroepithelial regions stimulate the vestibular nerve (CN VIII) and provide information about balance. The utricle and saccule as well as the semicircular ducts are filled with endolymph.



WARD TIP

The stimulating electrode in a cochlear implant is placed in the scala tympani.

Physiology

Evaluation of hearing

Sound waves can reach the cochlea in two ways:

- **Air conduction,** using the ear canal, TM, and middle ear structures. Pathology at any of these levels can decrease air conduction.
- **Bone conduction,** vibrating the temporal bone and therefore the cochlea, which lies within it, bypassing the external and middle ear structures; it depends on the integrity of cochlear structures, CN VIII, and central auditory pathways.

Causes of **conductive** hearing loss (CHL):

- Obstruction of external auditory meatus by cerumen, foreign body, or debris.
- Swelling of the EAC, neoplasms, or stenosis of the canal.
- Perforation of the TM.
- Ossicular discontinuity.
- Otosclerosis.
- Fluid, scarring, or neoplasms of the middle ear.

Causes of **sensorineural** hearing loss (SNHL):

- Damage to the hair cells of the organ of Corti by intense noise, barotrauma, viral infections (e.g., human herpesvirus), or ototoxic drugs (e.g., aminoglycosides).
- Presbycusis (normal hearing deterioration with aging).
- Iatrogenic.
- Temporal bone fractures.
- Meningitis.
- Cochlear otosclerosis.
- Ménière's disease (see later discussion).
- Acoustic neuromas (schwannomas).

Functional Testing

- **Weber test:** A 512-Hz fork is placed on the skull vertex; the patient is asked to which side the tone lateralizes. In CHL, the sound lateralizes to the affected ear. In SNHL, the sound lateralizes to the unaffected ear.
- **Rinne test:** Compares the ability to hear by air conduction to that of bone conduction. A vibrating tuning fork is placed on the patient's mastoid process, and the patient is asked to indicate if they hear the sound and when it stops. The tuning fork is then placed at the EAC. Normally, sound is

WARD TIP

The right ear is referred to as AD, the left ear as AS, both as AU.

heard louder by air conduction than by bone, called a positive Rinne test, but with CHL, bone conduction is louder, and thus the Rinne test is negative. With SNHL, this test is normal, although technically both bone and air sound perceptions are reduced.

Pathology

Otitis Media

Acute otitis media (AOM), recurrent AOM, chronic otitis media.

Definition

- Inflammation of the lining of the middle ear cleft along with a bacterial infection.
- More common in children.
- Eustachian tube dysfunction and a preceding upper respiratory infection (URI) are the most documented etiologic factors precipitating AOM.
- Common bacterial pathogens include Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis.

Signs and Symptoms

Otalgia, ear pulling, and systemic symptoms including fever, anorexia, vomiting, and diarrhea.

Diagnosis

On otoscopic exam, the TM appears to be bulging, red, and immobile on pneumatic otoscopy.

Treatment

- Symptom control and analgesia, antibiotic therapy, and prevention of complications.
- For cases of recurrent AOM (more than three episodes of AOM in a 6- to 12-month period) antibiotic prophylaxis is recommended.
- A myringotomy with or without tympanostomy tubes may be recommended if the AOM is not responsive to antibiotics.

Complications

Complications of otitis media include TM perforation, hearing loss, and infection or inflammation of the temporal bone or intracranial cavity.

Otitis Media with Effusion (OME)

Definition

Fluid collection in the middle ear cleft without the pain and fever associated with AOM.

Signs and Symptoms

Children usually present with hearing loss and speech delay.

Diagnosis

Otoscopic examination reveals decreased TM mobility, opacity, and, occasionally, evidence of an air–liquid interface behind the TM (air bubbles).

Treatment

- There is a clear indication for treatment of children with chronic OME with **myringotomy and tympanostomy tubes** if:
  - There is evidence of change significantly decreased hearing to "conductive hearing loss."

- The child is at high risk for speech and language delay (craniofacial disorders, cleft palate).
- The child has a current delay.
- There are recurrent bouts of AOM superimposed on OME.
- There is evidence of structural changes to the TM.

- An adeniodectomy may be performed simultaneously in select patients to improve eustachian tube function.

otitis Externa

Definition

- Generalized inflammation involving the EAC skin.
- Can be infectious or eczematous.
- Organisms include hemolytic Streptococcus, Candida, Staphylococcus, and Pseudomonas.
- **Malignant otitis externa** in immunocompromised patients (e.g., elderly diabetics) is usually caused by Pseudomonas spp. and can have more extensive complications (e.g., osteomyelitis).

Signs and Symptoms

- Ear pain, redness, tenderness, weeping, and otorrhea.
- It is often difficult to examine the ear due to pain.

treatment

- Analgesia to control the pain.
- Topical antibiotics + /– topical steroid cream.
- Systemic antibiotics for severe cases only.

Cholesteatoma

Definition

- Benign but invasive tumor of the middle ear/mastoid caused by overgrowth of displaced keratinizing epithelium.
- Can be **acquired** or **congenital.**

Signs and Symptoms

- Foul otorrhea, aural fullness or pressure, hearing loss.
- May cause CHL, SNHL, or CN VII injury or have intracranial extension.

Diagnosis

- Diagnosis is made by noting an area of retraction filled with debris on otoscopic examination.
- Evaluation with computed tomography (CT) scan and audiologic testing.

treatment

Surgery is the definitive treatment, necessary to eradicate disease and prevent recurrence. It is sometimes performed in two stages to allow assessment for residual disease in the mastoid and middle ear (i.e., second-look procedure).

acoustic Neuroma (Vestibular schwannoma)

Definition

- Benign tumor that arises in the cerebellopontine angle of the internal auditory canal (IAC).
- Associated with neurofibromatosis types I and II.

Signs and Symptoms

- The most common symptom is progressive asymmetric SNHL, followed by tinnitus and balance disturbance.
- Slow growing and nonmetastatic. They do, however, compress adjacent tissue as they grow, usually the vestibular and cochlear nerve within the IAC, but can compress the brain stem and facial nerve once they outgrow that space.

EXAM TIP

Classic time frames for vertigo episodes:
Minutes: BPPV
Hours: Ménière's disease
Days: Labyrinthitis

Diagnosis

Audiogram shows unilateral SNHL.

treatment

- Surgery or radiation therapy (RT): Surgical approaches include translabyrinthine, middle cranial fossa, and retrosigmoid/suboccipital.

WARD TIP

Extracranial branches of the facial nerve:
The **Z**ebra **B**it **M**y **C**ookie
Temporal
Zygomatic
Buccal
Marginal mandibular
Cervical
The posterior auricular branch comes off of the facial nerve before it splits into these five branches.

The Facial Nerve

The facial nerve innervates the muscles of facial expression; the digastric, stylohyoid, and stapedius muscles; as well as sensation to the anterior two thirds of the tongue and autonomic fibers for taste and to the salivary glands.

- **The circuitous path that the nerve takes has six segments:** Intracranial, meatal, labyrinthine, tympanic, mastoid, and extratemporal.
- It courses through the parotid gland on its way, and splits into five portions that innervate the face: Temporal, zygomatic, buccal, marginal mandibular, and cervical.

Facial nerve Paralysis

- Trauma.
- Iatrogenic injury.
- Injury related to temporal bone fractures.
- Viral infection (herpes zoster oticus [Ramsay Hunt syndrome]).
- Lyme disease.
- Bacterial infection (malignant otitis externa).
- Systemic disease (Guillain–Barré syndrome, mononucleosis, sarcoidosis).

Idiopathic Facial Nerve Paralysis: Bell's Palsy

Definition

- The most common form of paralysis.
- Ten percent of patients experience recurrent paralysis.
- Although the etiology is idiopathic, some suggest that the paralysis may result from a cycle of ischemic/inflammatory events of the nerve in the bony canal.
- There is no degeneration of the nerve.

WARD TIP

Lesions of the upper motor neuron tract of the facial nerve spare the forehead, as the upper motor neurons innervate the face bilaterally. Lesions of the lower motor neuron (past the geniculate ganglion) involve the entire ipsilateral side of the face.

granulomatosis with Polyangiitis (Wegener's granulomatosis)

Signs and Symptoms

- Occurs as a unilateral facial weakness of sudden onset that resolves spontaneously.
- There should be no signs of concurrent central nervous system (CNS) or ear disease.

- Sixty percent of patients experience a viral prodrome, and 60% experience facial, neck, or ear numbness or pain prior to the palsy.
- Symptoms usually improve within about 6 months, completely by 12 months.

Diagnosis

Evaluation includes a history and physical, an audiogram, and electrophysiologic testing of the nerve. If thought to be Bell's palsy and not due to some other cause, radiologic imaging is not recommended.

Treatment

- A course of steroids early in the disease process help shorten recovery time and serve as an analgesic.
- Surgical intervention with decompression of the nerve remains controversial.

WARD TIP

The scale used to measure facial nerve function is called the **House–Brackmann scale**. A score of 1 indicates complete function; 6 indicates complete loss of function.

Nose

Anatomy

- **External structure:** Paired nasal bones, midline septum, paired upper and lower lateral cartilages, soft tissue.
- **Internal structure:** The nasal cavity opens to the face through the nares (nostrils). Its axis is at a right angle to the face. Posteriorly, the choana communicates with the nasopharynx.
  - The internal nose is divided into two sides, bound medially by the nasal septum, composed of multiple cartilages and bony structures.
    - The septum is covered by perichondrium and periosteum, which is then covered by nasal mucosa (respiratory epithelium).
  - The lateral border of the nasal cavities consists of three bony ridges called turbinates, which each cover an area called a meatus. Each meatus contains ostia, which drain the paranasal sinuses. The superior and middle turbinates are parts of the ethmoid bone; the inferior turbinate is an independent bone.
- Four paranasal sinuses: Air spaces in the bony structure of the face that communicate with the nasal cavity via various ostia.
  - Paired maxillary sinuses laterally.
  - Ethmoids and sphenoids superoposteriorly.
  - Frontal sinuses superiorly.

Structure	Opening
Sphenoethmoidal recess	Sphenoid sinus
Superior meatus	Posterior ethmoidal air cells
Middle meatus	Frontal sinus, maxillary sinus, middle ethmoidal air cells, anterior ethmoidal air cells—osteomeatal complex
Inferior meatus	Nasolacrimal duct

WARD TIP

Only the maxillary and ethmoid sinuses are present at birth.

Blood supply

Both the internal carotid system, via the anterior and posterior ethmoidal arteries, and the external carotid system, via the internal maxillary/sphenopalatine artery, supply the nasal cavity and paranasal sinuses. The facial artery also supplies the anterior nose.

Nerve supply

Sympathetic autonomic innervation has a vasoconstrictive effect; **parasympathetic** fibers function in vasomotor stimulation.

Physiology

- **Nasal respiration:** Humidification, warming, and filtration of inhaled air and substances.
- **Olfaction.**
- **Phonation:** Normal voice depends on contribution of nasal resonance; disorders can give rise to hypo- or hypernasal speech.

Evaluation of the Nasal Cavity and Sinuses

- **Anterior rhinoscopy:** Using a nasal speculum. Allows visualization of the anterior portion of the nose, septum, and turbinates.
- **Nasal endoscopy:** Using a flexible or rigid scope. Provides diagnostic information, with attention to color, edema, discharge of nasal mucosa; allows for visualization of masses or abnormalities. Also allows for visualization of the nasopharynx, oropharynx, and hypopharynx/larynx.
- **Imaging:** Plain radiographic imaging of sinuses is of limited value. CT imaging has better resolution of structures, can evaluate for mucosal disease, provides a good "road map" for surgery. Can evaluate for septal deviation, polyps, masses, the osteomeatal complex, ethmoid air cells.

WARD TIP

The nasal cavity is usually decongested and topically anesthetized prior to endoscopy (Afrin and lidocaine).

Sinus Disease

Acute Bacterial Rhinosinusitis

Definition

- Infection of the paranasal sinuses/nasal mucosa, usually preceded by a URI, allergy, trauma, or dental infection.
- Impaired immune function predisposes to recurrent disease.
- Usually bilateral.
- Causative organisms include S. pneumoniae, H. influenzae, and M. catarrhalis.

Signs and Symptoms

Diagnosed after persistence of viral infection beyond 10 days, with severe or worsening symptoms, including maxillofacial pain, fever, dental pain, otalgia, malaise, and increased nasal drainage.

Treatment

- Medical management is with antibiotic therapy. Middle meatus culture or maxillary sinus aspiration can guide antibiotic treatment.
- Adjunctively, relief is provided with nasal saline irrigation, systemic decongestants, topical decongestants, and antihistamines if there is an allergic component.
- For treatment failure, a more comprehensive diagnostic workup is warranted, including imaging.

WARD TIP

Topical decongestants must be used with caution. If used for more than 3 days, rebound congestion, or rhinitis medicamentosa develops, which causes worsening of symptoms.

Chronic Sinusitis

Definition

Defined by sinusitis symptoms for > 3 months.

Management

- Patient receives maximal antibiotic therapy for 4–6 weeks prior to imaging studies, which often demonstrates anatomic variants predisposing the patients to osteomeatal compromise. Medical therapy often fails, and surgical therapy is necessary.
- **Functional endoscopic sinus surgery (FESS)** is the procedure of choice to promote natural drainage and aeration of the sinuses by opening up the osteomeatal complex.

ePistAxis

Etiology

Local Causes

- Trauma: Nose picking, external trauma, dry mucosa (common in the winter months).
- Barometric pressure changes.
- Sinusitis, URI, allergy.
- Neoplasia.
- Septal perforations (with cocaine use).

Systemic Causes

- Systemic disease: Hypertension, renal disease, hepatic failure, heavy alcohol use, granulomatosis with polyangiitis (aka Wegener's).
- Hematologic disease: Hemophilia, coagulopathy, thrombocytopenia.
- Vascular lesions: Arteriosclerosis, hereditary hemorrhagic telangiectasia (Osler–Weber–Rendu disease).
- Medication related: Anticoagulants, NSAIDs.

Assessment and Treatment

- Identify anterior vs. posterior and left vs. right bleeds by routine nasal speculum examination.
- Manage bleed based on severity and location. Decongestant and topical anesthetics help decrease the bleeding and allow for better visualization.
  - Anterior bleeds can usually be controlled with local pressure, pinching the tip of the nose with or without silver nitrate or electrocautery. An anterior nasal pack can be used as well, absorbable vs. nonabsorbable packing material.
  - A posterior bleed may require more vigorous management—posterior packing. The bleeding source can be more easily identified by nasal endoscopy.
- Determine the etiology of the bleed based on history and physical exam (coagulopathy, trauma, neoplasia) and manage that as necessary.
- Failure of appropriately placed packing with continued bleeding may require surgical therapy or sphenopalatine, anterior ethmoidal, or posterior ethmoidal artery ligation.
- Selective arterial embolization by an interventional radiologist is extremely effective as well, however, does carry a low risk for cerebral emboli.

WARD TIP

Ninety-five percent of bleeding occurs in the anterior nasal cavity, and ~90% of those are from Kiesselbach's plexus, a confluence of vessels at the anterior septum. These vessels are prominent and most subject to drying, mechanical trauma, and exposure to irritants.

WARD TIP

When dealing with posterior nasal bleeding, hemostasis can be achieved by inserting a Foley catheter into the patient's nose, inflating the balloon, and applying traction.

EXAM TIP

Nasal polyps in an infant/young child should raise suspicion of cystic fibrosis—do sweat chloride test.

nAsAl mAsses

Endoscopic examination, radiologic imaging, and surgical biopsy/removal are the mainstay of diagnosis and treatment.

Benign Masses

- **Inflammatory polyps:**
  - Treatment of choice is topical corticosteroids.
  - Systemic steroids for severe cases.
  - Surgical options for disease refractory to medical management include nasal polypectomy and FESS.
- **Antrochoanal polyps:**
  - Treatment is surgical removal. (Most benign masses tend to recur despite surgical excision)

- **Juvenile nasopharyngeal angiof broma (JNA):**
  - Treatment is primarily surgical removal.
  - Preoperative embolization can be performed within 72 hours prior to surgery to reduce blood loss.
- **Inverting papilloma** (~50% of nasal respiratory papillomas):
  - A benign nasal tumor that arises from the respiratory mucosa, associated with human papillomavirus (HPV), strains 6 and 11.
  - It is called inverting because the epithelium usually invades surrounding stromal tissue.
  - Treatment is with surgical resection, usually endoscopically.
  - There is a 5–10% chance of malignant transformation to squamous cell carcinoma (SCC).

Malignancies

- **Carcinoma:**
  - Nasal carcinoma accounts for between 27% and 35% of head and neck cancer.
  - Exposure to irritants and chemicals is associated with specif c nasal carcinomas.
  - Treatment by en-bloc resection via a craniofacial approach is choice; however, due to extent and spread of the cancer, it is often limited.
  - RT is often used as an adjuvant, with chemotherapy for palliation.
- **Lymphoma:**
  - The most common nonepithelial malignancy of the nose.
  - Usually associated with Epstein–Barr virus (EBV); more common in Asia.
  - Radiologic evaluation demonstrates opacif cation of the sinuses, bony erosion, and occasionally a mass.
  - Fresh biopsy sections need to be sent during surgical removal in order to identify the cancer.
  - Combined RT and chemotherapy is the preferred method of treatment, as there is a risk of distant recurrence with RT alone.
- **Melanoma:**
  - Nasal melanoma accounts for ~1% of all melanomas.
  - Patients are usually over the age of 60.
  - On nasal endoscopy, the lesions may appear as a benign polyp, or a dark, fungating neoplasm.
  - Treatment is by surgical resection, with a 5-year survival of about 30%.

Oral Cavity and Oropharynx

An At o my

- The oral cavity begins at the vermillion border of the lips, ends at the junction of the soft and hard palate superiorly and at the circumvallate papillae of the tongue.
- It includes the lips, buccal mucosa, superior and inferior alveolar ridges, part of the tongue, hard palate, and floor of the mouth.
- The salivary ducts terminate into the oral cavity, including the parotid Stensen's ducts lateral to the second maxillary molars, the submandibular Wharton's duct onto the floor of mouth, along with multiple sublingual duct orif ces that drain into the floor of mouth as well.
- The tongue is included in the oral cavity; the frenulum is a fold of mucosa that is anteriorly attached to the tongue that attaches it to the floor of mouth mucosa.

- The oropharynx begins at the junction of hard and soft palate superiorly and the circumvallate papillae on the tongue, and extends to the valeculae.
- The oropharynx contains a ring of lymphoid tissue called Waldeyer's ring. It consists of the adenoids superoposteriorly, the palatine tonsils laterally, and the lingual tonsil at the base of the tongue.

Blood supply

- The palate is supplied by small branches of the descending palatine artery, a branch of the maxillary artery.
- Vascular supply to tongue is via the lingual artery (a branch of the external carotid) and respective vein.
- The tonsils are supplied by the tonsillar and ascending palatine branches of the facial artery, by a branch of the lingual artery, by the ascending pharyngeal artery, and the lesser descending palatine branch of the maxillary artery.

Nerve supply

- The anterior two thirds of the tongue has somatic sensory innervation from CN V_3, and taste is via the chorda tympani (CN VII). The posterior one third of the tongue is innervated by CN IX (gag reflex) for sensory and taste.
- The muscles of the pharynx are innervated by CN X, except for the tensor veli palatini, which is innervated by CN V_3, and the stylopharyngeus, which is innervated by CN IX. Sensory innervation to the pharynx is by CN V_2, IX, and X.
- Sensory innervation to the oral cavity is via V_3.

Physiology

- The primary function of the oral cavity and oropharynx is mastication of food and food delivery to more distal structures.
- Saliva in the mouth lubricates and begins to digest the food.
- The tongue, lips, muscles, and palate move the bolus of food posteriorly into the oropharynx and then down to the esophagus.
- Once the bolus reaches the oropharynx, voluntary control of swallowing is switched to involuntary control to propel the bolus quickly past the closed glottis to the esophagus.
- During this time, the nasopharynx closes with palate elevation, and respiration must cease.

Peritonsillar abscess

Definition

- Located deep to the tonsillar capsule, usually at the superior pole.
- May be preceded by a pharyngitis.

Signs and Symptoms

Presents with fever, deviation of the tonsil and uvula, pain, soft palate swelling, trismus, and a hot potato voice.

Diagnosis

Clinical examination.

Complications

- Parapharyngeal abscesses if the infection spreads beyond the superior constrictor muscle, bacteremia, endocarditis, mediastinitis, airway obstruction, and aspiration pneumonia.

treatment

Needle drainage, incision and drainage, or tonsillectomy.

WARD TIP

Indications for tonsillectomy: Recurrent tonsillitis (6/year × 1 year, 5/year × 2 years, 3/year × 3 years), upper airway obstruction due to tonsillar hypertrophy (obstructive sleep apnea), recurrent peritonsillar abscess, malignancy.

Ranula

Definition

- A mucocele of the sublingual gland that presents as a cystic mass on the floor of mouth.
- It is called a "plunging ranula" when it penetrates the mylohyoid muscle, and presents as a soft submental neck mass.

Treatment

Excision should include the entire sublingual gland to prevent recurrence, with care taken dissecting around the lingual nerve and Wharton's duct.

Oral Malignancies

Oral Cavity Carcinoma

Definition

- Carcinoma of the lip, tongue, floor of mouth, gums, palate.
- Risk factors include tobacco smoke, pipes, betel nut chewing, alcohol, and sunlight exposure (cancer of the lip).
- Mostly SCC.

Signs and Symptoms

May present with leukoplakia or erythroplakia, mass or ulceration, or as symptoms due to the invasion of other structures (loose teeth, trismus).

Diagnosis

Assessment involves a biopsy, usually in the OR, along with panendoscopy to look for a second primary tumor. Patients also have a CXR and adjunctive imaging to assess the neck and evaluate extent of invasion. Staging with tumor-node-metastases (TNM) system.

Treatment

- Early stages are usually treated surgically.
- When advanced, combined modalities are used.
- The oral cavity has a rich lymphatic supply, so elective neck dissection is often recommended.
- Therapeutic neck dissection is performed when there is clinically apparent nodal disease.

Oropharyngeal Carcinoma

Definition

- Ninety percent SCC, keratizing or nonkeratizing (often with submucosal spread).
- Lymphocytic malignancies include lymphoma of the palatine tonsil and base of tongue. May be the first symptom of a systemic lymphoma.

Signs and Symptoms

Jugulodigastric and submental lymph node groups are most commonly affected.

Diagnosis

- Physical examination includes careful assessment of the pharynx and larynx, as second primary tumors are common.
- Use bimanual palpation (especially for base of tongue), nasal endoscopy, and panendoscopy under sedation.

treatment

- Treatment by RT is preferred, resulting in less morbidity and functional disturbance.
- Combined modalities, including surgery, chemotherapy, and RT are sometimes used as well, depending on where the lesion is (tonsil vs. base of tongue).
- Patients have an increased risk for distant metastasis; large bulky tumors and nodes should be followed closely and carefully for signs of local, regional, and distant recurrence.

Larynx and Hypopharynx

Anatomy

- Cartilaginous skeleton includes the cricoid, thyroid, arytenoids, corniculate, and cuneiform cartilages.
- The cricoid cartilage forms a complete ring.
- Superiorly, the hyoid bone forms an important framework for the larynx below it, as it is a major point of attachment for the extrinsic muscles of the larynx (see Figures 22-2 and 22-3).
- The laryngeal cavity is divided by the true vocal cords (TVCs):
  - Above them is the **supraglottis,** from the tip of the epiglottis to the apex of the ventricle, halfway between the true and false vocal cords. It includes the epiglottis, arytenoids, aryepiglottic folds, and false vocal cords.
  - The **glottis** is composed of the vocal folds—the middle of the laryngeal ventricle to 1 cm below the vocal folds.
  - The subglottis begins 1 cm below the TVC to the inferior edge of the cricoid cartilage.
- Muscle groups of the larynx are described as either:
  - **Extrinsic:** Depressors and elevators of the larynx, including the sternohyoid, thyrohyoid, omohyoid, geniohyoid, digastric, mylohyoid, and stylohyoid, innervated by cervical nerves, CN V, and CN VII.
  - **Accessory:** Pharyngeal constrictor muscles.
  - **Intrinsic:** Thyroarytenoid, thyroepiglottic, aryepiglottic; muscles of the arytenoids cartilage including the interarytenoid, posterior cricoarytenoid, and lateral cricoarytenoid muscles, which are responsible for abducting and adducting the vocal cords; and the cricothyroid muscle.

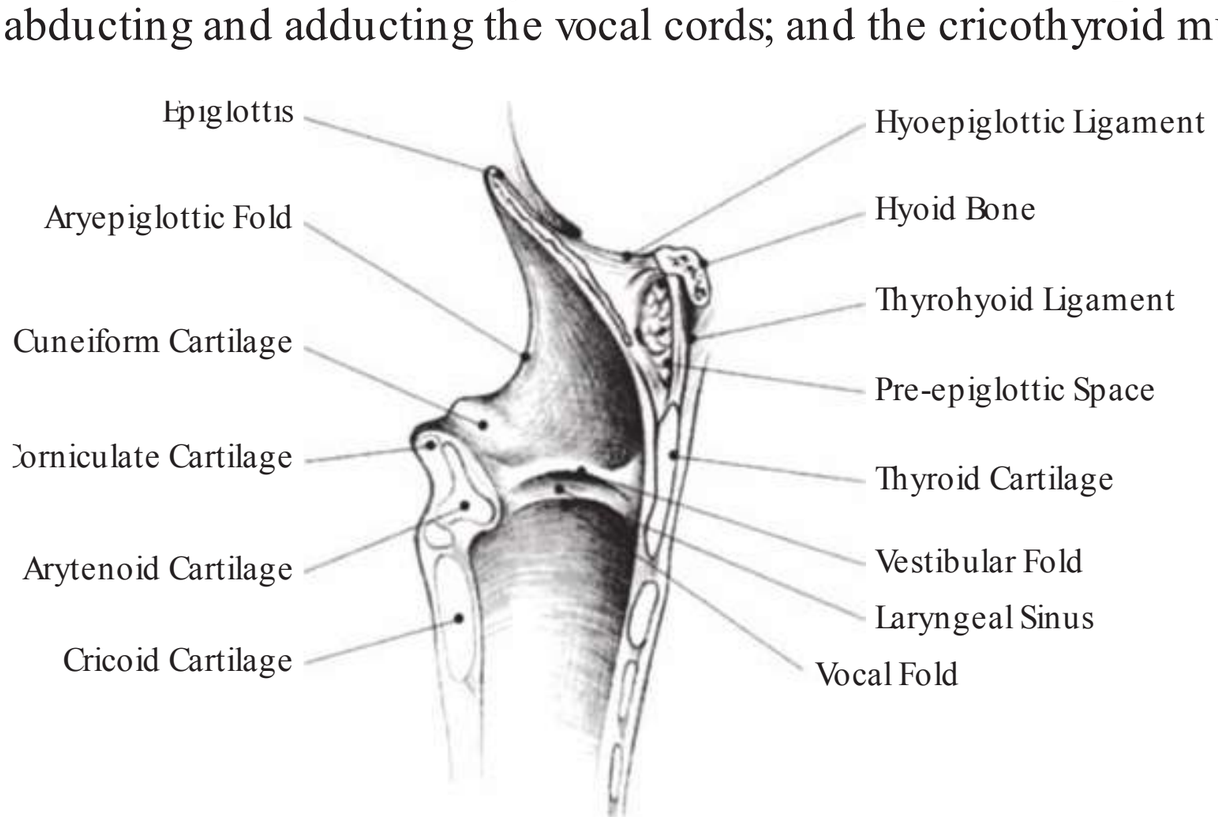


FIgurE 22-2. sagittal section of the larynx demonstrating anatomic divisions of the larynx. (Reproduced, with permission, from Lee KJ. Essential Otolaryngology: Head & Neck Surgery. 8th ed. New York: McGraw-Hill; 2003: 597.)

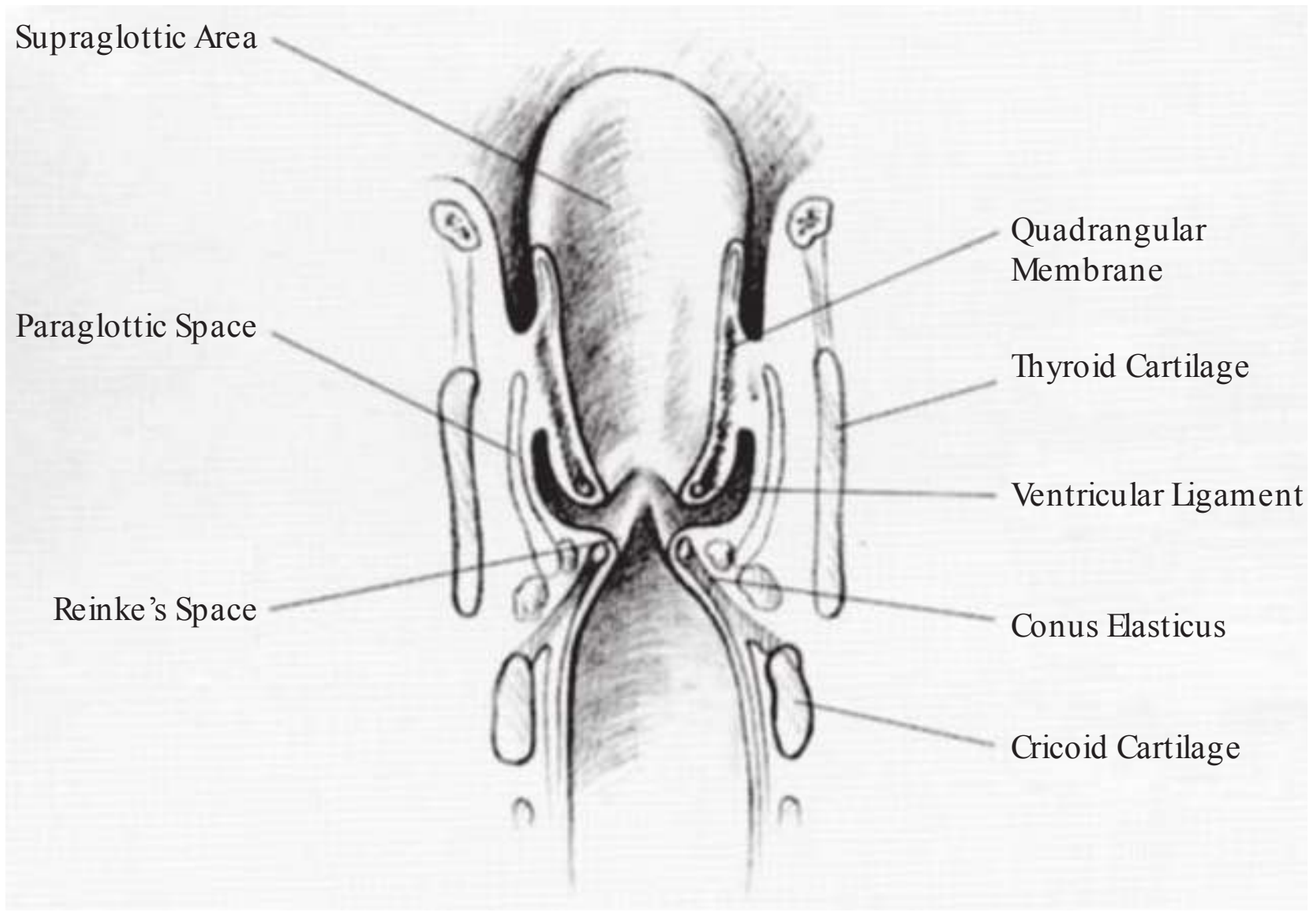


FIgurE 22-3. Coronal section of the larynx illustrating barriers to the spread of carcinoma. (Reproduced, with permission, from Lee KJ. Essential Otolaryngology: Head & Neck Surgery. 8th ed. New York: McGraw-Hill; 2003: 598.)

WARD TIP

An emergency surgical airway, cricothyroidotomy is created by puncturing through the cricothyroid membrane, situated anteriorly, between the thyroid cartilage and the cricoid cartilage.

Lymphatic drainage

- Drain mainly to the deep cervical group.
- The vocal cords themselves contain very few lymphatic channels, however the supra- and subglottic areas contain extensive lymphatic drainage (important in metastatic tumor spread).

Nerve supply

Two branches of the vagus nerve (CN X) supply the larynx:

- Superior laryngeal nerve: Divides into the external and internal branches.
  - **External:** Supplies motor fibers to the cricothyroid muscle only.
  - **Internal:** Sensory innervation to all areas of the larynx above the glottis.
- **Recurrent laryngeal nerve:** Due to embryonic development, the nerve descends into the thorax on the left side and passes under the aortic arch before it returns to the neck. Provides motor function to all of the intrinsic muscles of the larynx except for the cricothyroid muscle; sensory innervation to the laryngeal mucosa below the glottis.

Physiology

Functions of the larynx include:

- **Phonation:** The vocal cords act by vibrating. Intrinsic muscles determine the vibratory characteristics such as tension and contour of the vocal cords. The lateral cricoarytenoid and thyroarytenoid muscles adduct the cords, approximating the vocal processes and close the glottis during phonation; the posterior cricoarytenoid muscle primarily abducts the cords, thereby opening the glottis.
- **Protection of the respiratory tract:** The larynx acts as a sphincter—muscles elevate and close the larynx, determine epiglottis position, vocal cord adduction, cough reflex.
- **Respiration:** Muscular dilatation of the laryngeal aperture.

Evaluation of the Larynx

Indirect laryngoscopy:

- **Mirror examination:** Noninvasive but limited exam of the larynx, headlight illumination of a laryngeal mirror held up against the uvula, reflects an image of the larynx.
- **Flexible fiberoptic examination:** Through the nose.
- **Videostroboscopy:** A large-bore, rigid, 90° fiberoptic endoscope. A camera inside the endoscope continually flashes at a predetermined speed, allowing assessment of vocal fold symmetry, the mucosal wave, and subtle lesions.
- **Direct laryngoscopy:** Done in the OR under general anesthesia. A rigid laryngoscope is inserted; one can perform procedures through the scope (biopsy, laser).

WARD TIP

- Stridor: The audible result of turbulent airflow in the larynx/trachea.
- Inspiratory: Supraglottic pathology
- Expiratory: Tracheal pathology
- Biphasic: Glottic or subglottic pathology

Distinguished from **stertor,** which is generated by pharyngeal obstruction (snoring).

Benign Lesions of the Vocal Cords

Polyp

Definition

- A pedicled or sessile lesion that occurs on the vocal cords.
- They vary in size and appear as a smooth, glistening body.
- Can overhang the vocal cords, and may be evident on examination only when the patient is asked to cough and the polyp arises from the undersurface of the cords.

Signs and Symptoms

- Presents as long-standing hoarseness.
- Occasionally, they can be large and cause stridor and airway obstruction.

treatment

- Definitive treatment is by lifestyle modifications (smoking, voice abuse), endoscopic removal of the lesion, and stripping of the cords if lesions are bilateral.
- If airway obstruction occurs, tracheotomy or laser debulking of the lesions may be necessary.

Nodule ("singer's Nodules")

Definition

- Localized traumatic laryngitis caused by vocal overuse (screaming, harsh talking, faulty singing technique), allergy, URI, sinusitis, smoking, and alcohol.
- Site of injury is usually the epithelium and basement membrane of the vocal cords.

Signs and Symptoms

- Usually presents as hoarseness.
- On exam, nodules are commonly found at the junction of the anterior and middle third of the vocal cords, the area of maximum vibration of the cords.
- Usually bilateral.

treatment

- Behavioral modification, vocal reeducation, voice rest, and therapy.
- Microlaryngoscopic excision if nonresolving.

WARD TIP

Laryngeal Foreign Bodies

Most commonly food related. Obstruction is usually relieved by the normal reflexes. General anesthesia and rigid bronchoscopy is recommended for removal of the foreign body. If the patient becomes completely obstructed during the removal, the foreign body should be pushed further into the bronchus so that the contralateral lung can be ventilated.

Papilloma

Definition

- The most common benign tumor of the larynx.
- Bimodal age distribution.
- Causative agent thought to be HPV. Certain subtypes of the virus can undergo malignant transformation.
- Can involve any region of the larynx and trachea, as well as the lungs.

Signs and Symptoms

Aphonia or a weak cry in infants, dyspnea and stridor, hoarseness.

treatment

Treatment includes CO_2 laser excision, microdebridement, therapy with interferon, intralesional cidofovir injection, topical application of mitomycin C (an antineoplastic).

laryngeal cancer

definition

- Malignant tumors of the larynx and pharynx are usually SCC.
- The most common site for carcinoma in the upper aerodigestive tract.
- As there is an extensive lymphatic supply to the supra- and subglottic regions of the larynx, there is well-defined metastasis to the lymph nodes of the jugular chain or paratracheal regions. The exception to this rule is glottic carcinoma, which presents earlier and has a small likelihood of lymphatic metastasis (owing to the poor lymphatic drainage of the glottis).
- Direct spread and invasion by tumors can occur as well.
- The etiologic factors for most laryngeal SCC are tobacco smoke and alcohol exposure, which act synergistically.

signs and symptoms

- The presentation of laryngeal cancer is usually progressive hoarseness due to interference with the vocal cords.
- Other symptoms include dyspnea, dysphagia from pharyngeal involvement, and referred ear pain (an ominous sign).

diagnosis

- Laryngoscopy must be done on a patient who has a persistent hoarse voice for > 2 weeks if over 40, and after 3–4 weeks if younger, assessing for asymmetry and the extent of any lesions.
- Tumors of the cords appear to be raised and warty in appearance; those of the supraglottis are usually exophytic.
- Vocal cord mobility must be noted.
- The neck is examined for metastatic lymphadenopathy.
- Diagnosis can occur after assessment of the larynx, pharynx, and oral cavity by direct microlaryngoscopy under general anesthesia.
- Depth of invasion and extent of the lesion need to be determined to stage the lesion (TNM classification) and guide treatment.
- Biopsies are taken.

Treatment

- Depends on stage and patient characteristics.
- It is either definitive removal or palliative.



WARD TIP

Tracheotomy vs. Tracheostomy vs. Laryngectomy

Tracheotomy: A temporary opening in the trachea; indicated for mechanical obstruction at or above the larynx, difficulty with clearing excessive secretions, airway protection during radical neck surgery; prolonged need for mechanical ventilation. These patients have intact oral airways and can be intubated through the orotracheal or tracheal approach (tracheostomy: more permanent).

Laryngectomy: A radical procedure to remove the larynx; indicated for advanced and recurrent laryngeal cancer. The trachea is rerouted to terminate at a stoma in the neck, and there is no longer patency between the oral cavity and the trachea. A patient like this **cannot** be intubated orally—must be intubated through their tracheal stoma.

- Early tumors are treated by a single modality, usually radiotherapy, and more complicated cases often with a laryngectomy (partial or total depending on the disease state) and postoperative RT.
- Options are available for voice reconstruction (a tracheoesophageal one-way valve) and return of swallowing that allow these patients to have a good quality of life.

Salivary Glands

Anatomy

Parotid

- Largest salivary gland; enclosed by deep cervical fascia. Composed of both deep and superficial lobes, separated by a plane of the facial nerve.
- Drains lateral to the second upper molar via Stensen's duct.
- The facial nerve passes through the gland, and divides into its five branches.
- Parasympathetic supply originates in the inferior salivatory nucleus, travels with CN IX to Jacobsen's nerve to the otic ganglion; synapses and fibers are carried on the auriculotemporal nerve of CN V_3.
- Sympathetic fibers come from the superior thoracic nerves and synapse with the superior cervical ganglion. They travel via arterial plexuses and sensory nerves to the gland.



WARD TIP

The facial nerve separates the superficial lobe of the parotid from the deep lobe.

Submandibular

- Drains into the mouth via Wharton's duct, which courses between the sublingual gland and hyoglossus muscle; opens through a small opening lateral to the frenulum on the floor of mouth.
- Parasympathetic supply originates in the superior salivatory nucleus, travels via nervus intermedius of CN VII to the chorda tympani, which then joins the lingual nerve to the submandibular ganglion. Fibers synapse there and travel to the gland.
- Sympathetic supply is the same as for the parotid gland.

Sublingual

Smallest major salivary gland; lies in a submucosal location on the floor of mouth, and opens there through numerous small ducts called the ducts of Rivinius.

Minor salivary glands

Between 600 and 1,000 distributed all over the oral cavity and oropharynx, mostly on the hard and soft palate.

Physiology

- Parasympathetic stimulation increases saliva secretion; sympathetic slows it down.
- Saliva is high in potassium, low in sodium; it contains substances that begin the breakdown of food, to maintain and protect the oral cavity environment, and immunoglobulin A (IgA).
- Produce between 0.5 and 1.5 L of saliva/day.

Radiographic Evaluation

- CT/MRI is sometimes performed after injection of contrast medium directly into the ducts.
- Diagnostic ultrasound is also useful but has limitations.
- Fine-needle aspiration (FNA) for diagnosis of tumors vs. inflammatory lesions.

Benign/Systemic Disease

Sjögren's Syndrome

Definition

- An autoimmune connective tissue disorder that is often associated with rheumatoid arthritis and systemic lupus erythematosus (SLE).
- Patients affected are usually middle-aged, menopausal women.

Signs and Symptoms

Presents with keratoconjunctivitis sicca (dry, itchy eyes), xerostomia, taste changes, enlargement of salivary glands.

Diagnosis

The diagnosis is made clinically: Measurement of salivary flow rates and salivary gland biopsy showing lymphocytic infiltrate and acinar atrophy confirming suspicions, in addition to relevant rheumatologic tests.

Treatment

Treatment for xerostomia and dry eyes is symptomatic: Salivary and lacrimal substitutes.

Sialolithiasis

Definition

- Hydroxyapatite stones in the salivary glands/ducts.
- Most commonly affects the submandibular gland.
- More common in middle-aged men.

Signs and Symptoms

Presents with pain and swelling in the affected area, with symptoms worsening prior to eating.

Treatment

- If the stone is in Wharton's duct, it can be surgically removed transorally.
- If it is closer to the hilum of the gland, the gland may need to be removed.

Sialoadenitis

Definition

- Acute form is due to inflammation of the gland.
- Chronic form is a recurrent, painful enlargement of the gland, caused by salivary stasis.
- Occurs in debilitated and dehydrated patients, after major surgery, trauma, x-ray therapy (XRT).
- Infectious agent is typically Staphylococcus aureus.

Signs and Symptoms

Erythema, pain, swelling, and purulent ductal discharge.

Treatment

- Acute form: Rehydration, sialogogues, warm compresses, and antibiotics.
- Chronic form: Medication to stimulate secretions, massages of the gland, and hydration.

Mumps

Definition

Infection with paramyxovirus in children, usually with a history of inadequate immunizations.

Signs and Symptoms

Bilateral painful parotid swelling, trismus, and malaise, along with other systemic manifestations (orchitis, pancreatitis, encephalitis).

Diagnosis

Measurement of antibody titers.

Treatment

This condition is usually self-limited, so supportive symptomatic treatment is recommended.

Salivary gland Tumors

- FNA may be a useful tool in diagnosis, especially for malignant lesions. CT/MRI imaging in conjunction, especially when planning for surgery.

Benign tumors

Pleomorphic adenoma

- Most common benign tumor; slight predilection for middle-aged women.
- Myoepithelial cells are thought to be the cell of origin.
- There is morphologic diversity: The tumor can be mucoid, chondroid, osseus, or myxoid.
- Slow growing, painless swelling of the gland, usually in the posterior region of the parotid gland.
- Rarely undergoes malignant transformation to carcinoma ex pleomorphic.
- Treatment: Parotidectomy.
- Care must be taken to expose the facial nerve proximal to where it enters the gland, and to follow it forward, dissecting tumor off of the nerve with care to preserve function.

Monomorphic adenoma

- A benign tumor with features similar to pleomorphic adenoma, but with only one morphologic cell type present.
- Slow growing and solitary, most often in the parotid gland.

Warthin's Tumor

- AKA: Papillary cystadenoma lymphomatosum.
- Men > women (5:1); usually in older individuals.
- Often presents as a benign, painless, compressible, slow-growing mass. Can be bilateral in 10%.
- Treatment: Excision of the involved gland.

Oncocytoma

- Benign tumor, mostly in the parotid; accounts for < 1% salivary gland tumors.
- Slow growing and circumscribed, not encapsulated.



WARD TIP

Frey's syndrome: Gustatory sweating following parotid surgery, due to cross-reinnervation of the autonomic supply of the parotid gland, now to the overlying skin's sweat glands and blood vessels. Treat with an anticholinergic cream

Malignant tumors

Mucoepidermoid Carcinoma

- Most common malignant tumor of the salivary glands; derived from epithelial cells in the interlobar and intralobar epithelial cells of the gland.
- Can vary from low grade to highly malignant.
- Symptoms: Range from asymptomatic swelling (75%) to pain and facial nerve paralysis.
- Mostly affects the parotid.
- Most commonly induced by prior irradiation.
- Lymph node metastases are common in ~40% of patients.

Carcinoma Ex Pleomorphic

- Usually presents in patients who have undergone resection of a pleomorphic adenoma.
- Pathology shows remnants of benign mixed tumor.

Adenocarcinoma

- Twenty percent of salivary tumors and only 4% of parotid tumors are adenocarcinomas.
- Eighty percent of patients are asymptomatic, although 20% have facial nerve paralysis and 15% facial pain due to fixation of the tumor to underlying/overlying structures.
- Arises from the terminal tubules and intercalated or strained duct cells in the gland.
- Many varieties have been described, and are graded as low, intermediate, or high.

Adenoid Cystic Carcinoma

- Origin is the myoepithelial cell.
- Most common malignant tumor of the submandibular and minor salivary glands.
- Malignant but slow growing; most patients are asymptomatic on presentation even though a large percentage of those tumors are fixed to adjacent structures.
- Neurotropic tumor with early distant metastasis.

Acinic Cell Carcinoma

- Occurs exclusively in the parotid gland.
- Women > men.
- Pathologically defined by the presence of amyloid.
- Cell of origin in the serous acinar components and the intercalated duct cells.

Neck

Anatomy

- The neck is traditionally divided into anterior and posterior triangles (Figure 22-4).
- There are two main fascial planes (Figure 22-5):
  - **Superficial cervical fascia:** Encloses the platysma and muscles of facial expression. It begins at the zygoma of the face and extends inferiorly to the clavicle.

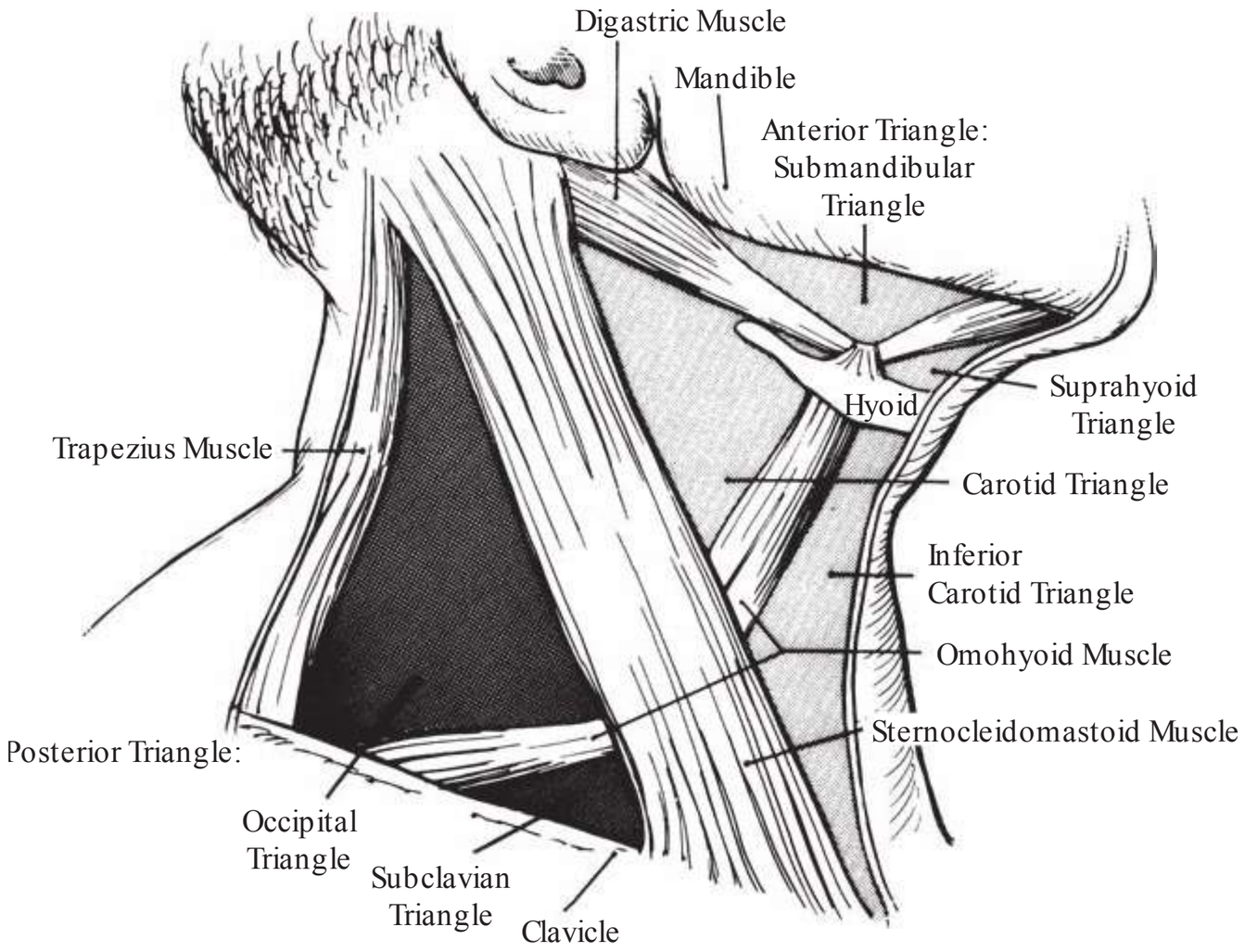


FIgurE 22-4. Triangles of the neck. (Reproduced, with permission, from Lee KJ. Essential Otolaryngology: Head &Neck Surgery. 8th ed. New York: McGraw-Hill; 2003: 423.)

- **Deep cervical fascia:** Composed of four layers:
  1. **Superf cial layer/investing fascia:** Encloses the trapezius, sternocleidomastoid (SCM), and strap muscles; submandibular and parotid glands; and the muscles of mastication. Stretches from the mandible to the clavicle.
  2. **Middle layer/visceral/pretracheal fascia:** Encloses the pharynx, larynx, trachea, and esophagus, thyroid/parathyroid glands, buccinator and constrictors, and the strap muscles. It goes from the skull base to the mediastinum. Posteriorly, this fascia forms a midline raphe that connects to the alar layer of the prevertebral fascia.
  3. **Deep layer/prevertebral fascia:** Encloses the paraspinous and cervical muscles; goes from skull base to the chest. Anteriorly, there are two layers to this fascia—the prevertebral layer lies anterior to the cervical vertebrae from the skull to the coccyx; anterior to that is the alar

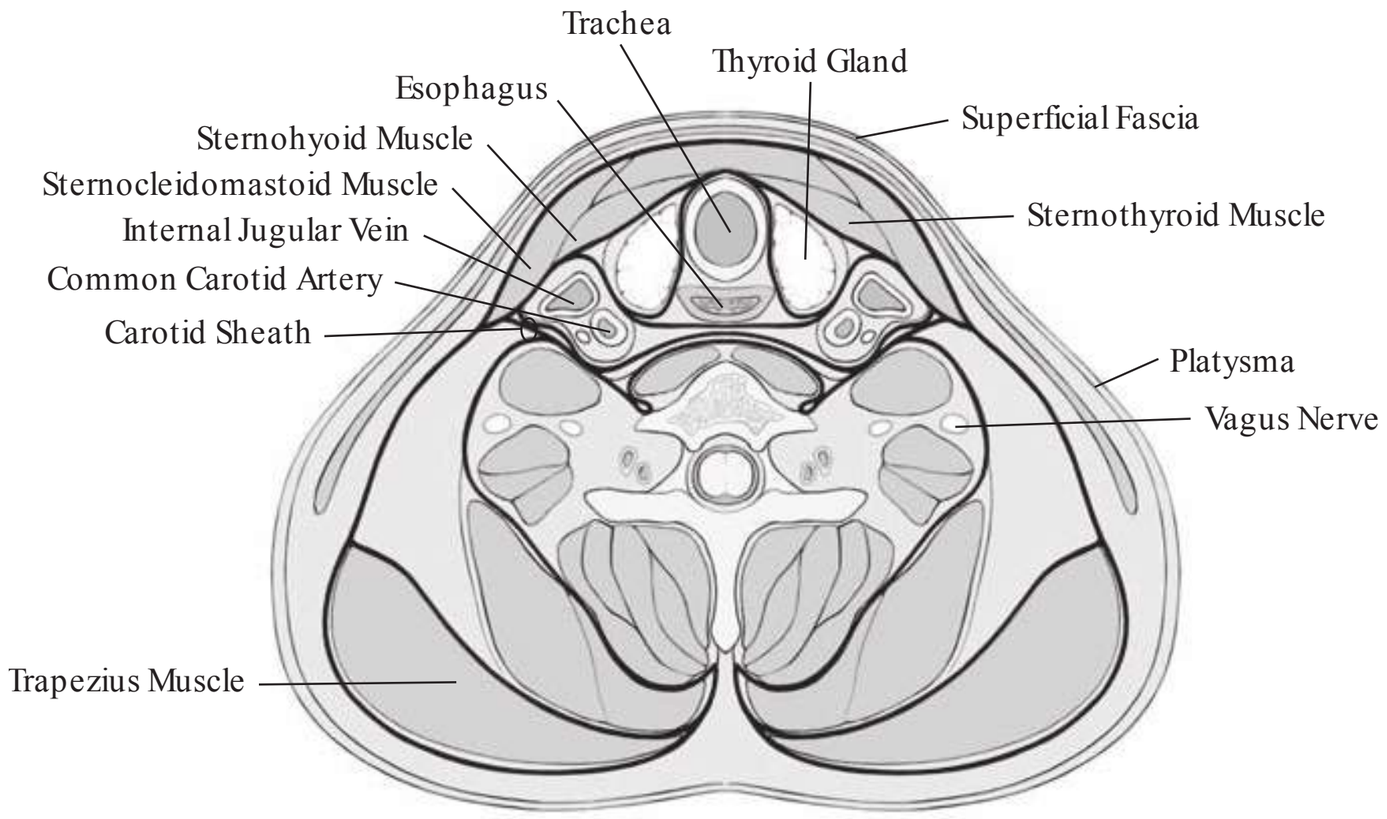


FIgurE 22-5. Fascial planes of the neck.

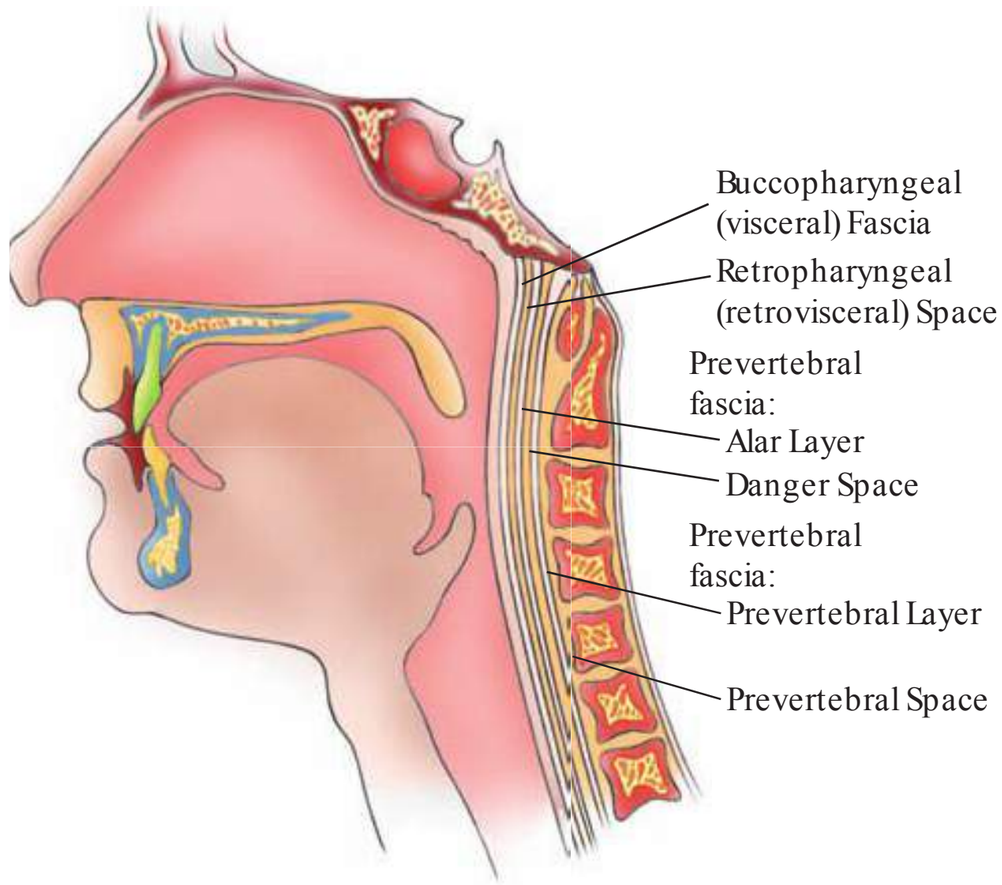


FIgurE 22-6. Fascial layers of the retrovisceral space.

layer, which extends from the base of skull to the mediastinum. The danger space lies between the alar and prevertebral layers. Anterior to that is the visceral/buccopharyngeal fascial layer of the middle fascia.

4. **Carotid sheath fascia:** Envelopes the carotid artery, internal jugular vein, CN X. Runs from base of skull to the thorax.

- The spaces formed by the neck fascial layers are potential spaces for an infection to extend to, seed, and spread (Figure 22-6).
- **Above the hyoid bone:**
  - **Parapharyngeal:** Infections spread from tonsils, pharynx, teeth, parotid gland, and extension from other spaces. Parotid involvement, trismus, fever, and sepsis common with an infection here. Extraoral approach for drainage.
  - **Submandibular.**
  - **Masticator:** Infected when molar teeth infection spreads.
  - **Parotid.**
  - **Peritonsillar:** Loose connective tissue that lies between the tonsillar capsule medially and the superior constrictor laterally. (See section on peritonsillar abscess earlier in this chapter.)
- **Below the hyoid bone:** Visceral space encloses the pharynx, esophagus, larynx, trachea, and thyroid. The prevertebral and retropharyngeal spaces lie posterior to it.
- **Entire length of the neck:**
  - **Retropharyngeal:** Anterior to the alar layer and posterior to the buccopharyngeal fascia/esophagus and pharynx, an infection from here can spread to the danger space. Contains lymph nodes and connective tissue. The greatest number of LN here are found in children < 4 years old, and accounts for a large number of retropharyngeal abscesses.
  - **Danger/alar space:** Between alar layer and the prevertebral layer of deep cervical fascia. Infection spreads to superior mediastinum.
  - **Prevertebral:** Infection can spread to the coccyx.
  - **Vascular:** Infection of the carotid sheath.

Vascular supply

Carotid and subclavian systems.

Nerve supply

To the skin: C2–C4.

Lymphatic drainage

- The neck has a rich lymphatic network, and lymphogenous drainage from different sites in the head and neck is highly predictable.
- Lymph node areas are divided into different levels, which become relevant when performing a neck dissection and staging nodal disease.

CONGENITAL DISEASES IN THE NECK

Branchial apparatus anomalies

- The head, neck, and related structures form embryologically from five branchial arches, grooves, and pouches (Table 22-1).

TABLE 22-1. The Five Branchial Arches

Arch	Nerve	Bones/Cartilage	Muscles/Vessels	Other
First arch	Mandibular CN V_3	Mandible, malleus, incus	Muscles of mastication, tensor tympani, anterior belly of digastric, tensor palatine	The pouch forms the middle ear cavity, and part of the tonsillar fossa and palatine fossa. The groove forms the EAC. A fistula would extend from the skin of the neck to the regions of the eustachian tube.
Second arch	Facial CN VII	Stapes, part of hyoid	Stapedius muscle, facial muscles, buccinators	The pouch forms the tonsillar fossa, palatine tonsil. A fistula would extend from the skin on the lower one third of the neck, anterior to the SCM, to the supratonsillar fossa. These are the **most common** fistulas.
Third arch	Glossopharyngeal CN IX	Part of hyoid bone	Stylopharyngeus, superior and middle constrictors, common and internal carotid artery	The pouch forms the thymus and inferior parathyroid gland
Fourth arch	Vagus CN X/superior laryngeal nerve	Thyroid and cuneiform cartilage	Inferior pharyngeal constrictor, cricopharyngeus, cricothyroid muscle, L aorta, and proximal R subclavian artery	The pouch forms the superior parathyroid glands, ultimobranchial body
Sixth arch	Vagus/CN X—recurrent laryngeal branch	Cricoid, arytenoids, corniculate, and tracheal cartilage	Intrinsic laryngeal muscles, inferior constrictor muscle, and ductus arteriosus	There is no associated pouch

- Anomalies occur when pouches persist as a branchial sinus or a branchial fistula develops between the pouch and groove. A cyst can also develop if part of a groove or pouch becomes separated from the surface and does not resorb and become prone to repeated infections; need to be excised completely.

Thyroglossal duct Cyst

embryology/Definition

- The thyroid develops from the foramen cecum at the base of the tongue and migrates down to the root of the neck along the thyroglossal duct.
- A remnant of the embryological migration becomes a thyroglossal duct cyst.

Signs and Symptoms

- Presents as a midline infrahyoid structure.
- The cyst can be at any level along the route of the duct, and usually moves with swallowing and protruding the tongue because of its attachment to the base of tongue.
- These cysts can become infected and drain cutaneously.

treatment

- Surgical excision of the gland remnant (Sistrunk procedure), provided that ultrasound investigation reveals normal thyroid gland.
- The procedure involves removing the ectopic gland, duct, and central portion of the hyoid bone to minimize the chances of recurrence.

Lymphatic Malformation

embryology/Definition

A malformation of the lymphatic system results in a multilocular neck mass filled with straw-colored fluid (formerly known as a cystic hygroma).

Signs and Symptoms

Usually presents at birth with extensive neck and facial swelling; may complicate the airway.

treatment

- Resection is indicated, both functionally and cosmetically.
- Instillation of a sclerosing agent has shown some promise as well.

WARD TIP

Although a neck abscess is usually suppuration of a reactive lymph node, investigation must be taken as to whether the node is infected with mononucleosis, tuberculosis (TB), or lymphoma. FNA of a TB abscess without excision can lead to a persistent draining sinus tract, and a lymphoma must be excised for diagnosis, not just needle biopsied.

infectious/inflammatory lesions of the neck

Ludwig's angina

Definition

- Acute cellulitis of the submandibular triangle deep to the mylohyoid muscle. An emergency! Risk of sepsis and airway compromise.
- Usually arises from an oral cavity infection.

Signs and Symptoms

- Triangle is bound by attachment of the deep cervical fascia to the mandible and hyoid; suppuration that builds up creates a lot of pressure and pain.
- Infection can track posteriorly and potentially cause laryngeal edema.

Treatment

- Aggressive treatment with IV antibiotics is necessary.
- Intubation or tracheostomy may be needed to protect the airway.
- If nonresolving, may need I&D.

Retropharyngeal Abscess

Definition

- Usually results from a suppurating lymph node in the retropharyngeal space.
- Can be secondary to a penetrating pharyngeal injury.
- They can traverse to the danger space and track down to the mediastinum.

Signs and Symptoms

Patients are ill and febrile, dehydrated, complain of dysphagia and pain, and may be stridulous.

Diagnosis

Lateral soft tissue radiography is helpful in diagnosis when there is marked swelling of the prevertebral tissues, and CT can be used to find the exact location of the abscess.

Treatment

Drain abscess; maintain airway with an endotracheal tube or a tracheostomy.



WARD TIP

Intubate (consider nasotracheal intubation) before excision of Ludwig's angina abscess if evidence of airway compromise.

Malignancy

Thyroid/Parathyroid Tumors

See Endocrine System chapter.

Lymphoma

Signs and Symptoms

- A disease of young and middle-aged adults.
- Usually presents with multiple, slow-growing, rubbery lymph nodes in the neck, which may be the only presenting symptom of the disease.
- Systemic symptoms, including fever and night sweats, imply a worse prognosis.

Diagnosis

Open biopsy of the node. Cellular architecture is important in both diagnosing Hodgkin's vs. non-Hodgkin's lymphoma and determining subtype of each.

Treatment

Depends on type and stage, and can include chemotherapy or RT.

Metastatic Lymphadenopathy

Signs and Symptoms

- Neck mass is the presenting sign of SCC in the head and neck or other anatomic location.
- Usually presents in older patients with a firm neck mass.

Diagnosis

- FNA helps differentiate between metastatic disease and other causes of chronic cervical lymphadenopathy (e.g., TB).
- Panendoscopy of the upper aerodigestive tract is useful for diagnosis.



WARD TIP

Neck dissection
radical: Removal of all levels of lymph nodes, the SCM, internal jugular vein, and CN XI.
Modified radical: A more selective procedure—removal of all levels of lymph nodes, but preservation of either SCM, internal jugular vein, or CN XI.
selective: All levels of lymph nodes are not removed.

WARD TIP

The unknown primary head and neck cancer: Identify based on lymph node presentation pattern and characteristic locations draining to lymphatic groups.

treatment

Dictated by the location of the primary tumor. A surgical dissection and removal of the neck nodes and associated structures is necessary to pathologically identify and treat the disease.

References

Lee KJ. Essential Otolaryngology: Head and Neck Surgery. 8th ed. New York: McGraw-Hill; 2003.

Burton M. Hall and Colman's Diseases *of* the Ear Nose and Throat. 15th ed. New York: Churchill Livingstone; 2000.

HIGH-YIELD FACTS IN

Neurosurgery

General Neuroanatomy 362
Cranial Foramina 362
Central Nervous System (CNS) Vasculature 362
major iCa BranCheS VisiBle with angiography 362
anterior CereBral artery (aCa) BranCheS 362
middle CereBral artery (mCa) BranCheS 362
VerteBral artery BranCheS 362
Basilar artery BranCheS 362
posterior CereBral artery BranCheS 362
arterial 363
Head Trauma 363
anatomy and physiology 363
Skull Fractures 365
linear (nondepressed) 365
Diffuse Intracranial Lesions 365
CereBral ConCuSSion 365
diFFuSe axonal injury (dai) 365
Focal Intracranial Lesions 366
CereBral Contusion 366
intraCereBral hemorrhage 366
epidural hematoma 366
SuBdural hematoma 366
Management of Mild to Moderate Head Trauma 367
Management of Severe Head Injury (GCS < 9) 368
hydroCephalus 368
CNS Tumors 370
general 370
presentation 371
Primary Brain Tumors 371
glial tumor 371
aStroCytoma 371
oligodendroglioma 371
ependymoma 371
meningioma 372
pituitary adenoma 372
neuroma 372
CraniopharyngioMa 372
hemangioBlaStoma 373
glomus tumors 373
Metastatic Tumors 373
imaging 373
eValuation 373
management 374
Spine 374
deFinitions 374
low BaCk pain 374
Spinal Trauma 374
general 374
anatomy 374
pathology and pathophysiology 374
neurogeniC ShoCk 375
Spinal ShoCk 375
Spinal Cord injuries 375
Spinal Cord Syndromes 376
CerViCal Spine FraCtures and diSloCations 377
thoraCiC Spine FraCtures 379
thoraColumBar junCtion and lumBar Spine FraCtures and diSloCations 379

General Neuroanatomy

Cranial Foramina

- **Optic canal:** Optic nerve and ophthalmic artery.
- **Superior orbital fissure:** Cranial nerves (CN) III, IV, VI, and V_1.
- **Foramen rotundum:** CN V_2.
- **Foramen ovale:** CN V_3.
- **Carotid canal:** Internal carotid artery.
- **Internal acoustic meatus:** CN VII and VIII.
- **Stylomastoid foramen:** CN VII and stylomastoid artery.
- **Jugular foramen:** Internal jugular vein and CN IX–XI.
- **Hypoglossal canal:** CN XII.
- **Foramen spinosum:** Middle meningeal artery and vein.
- **Foramen magnum:** Spinal cord, CN XI (spinal accessory), and vertebral, posterior, and anterior spinal arteries.

Central Nervous System (CNS) Vasculature

Internal carotid artery (ICA) does not produce branches until it enters the petrous bone, where it gives off geographically occult feeders to the middle and inner ear.

major iCa Branches Visible with angiography

Meningohypophyseal, inferolateral trunk, ophthalmic, posterior communicating, anterior choroidal, middle cerebral, anterior cerebral.

anterior Cerebral artery (aCa) Branches

Medial lenticulostriates, anterior communication, recurrent artery of Heubner, orbitofrontal, frontopolar, pericallosal, callosomarginal.

middle Cerebral artery (mCa) Branches

Lateral lenticulostriates, anterior temporal, posterior cerebral, ascending frontal, lateral orbitofrontal, precentral, central, anterior parietal, posterior parietal, angular.

Vertebral artery Branches

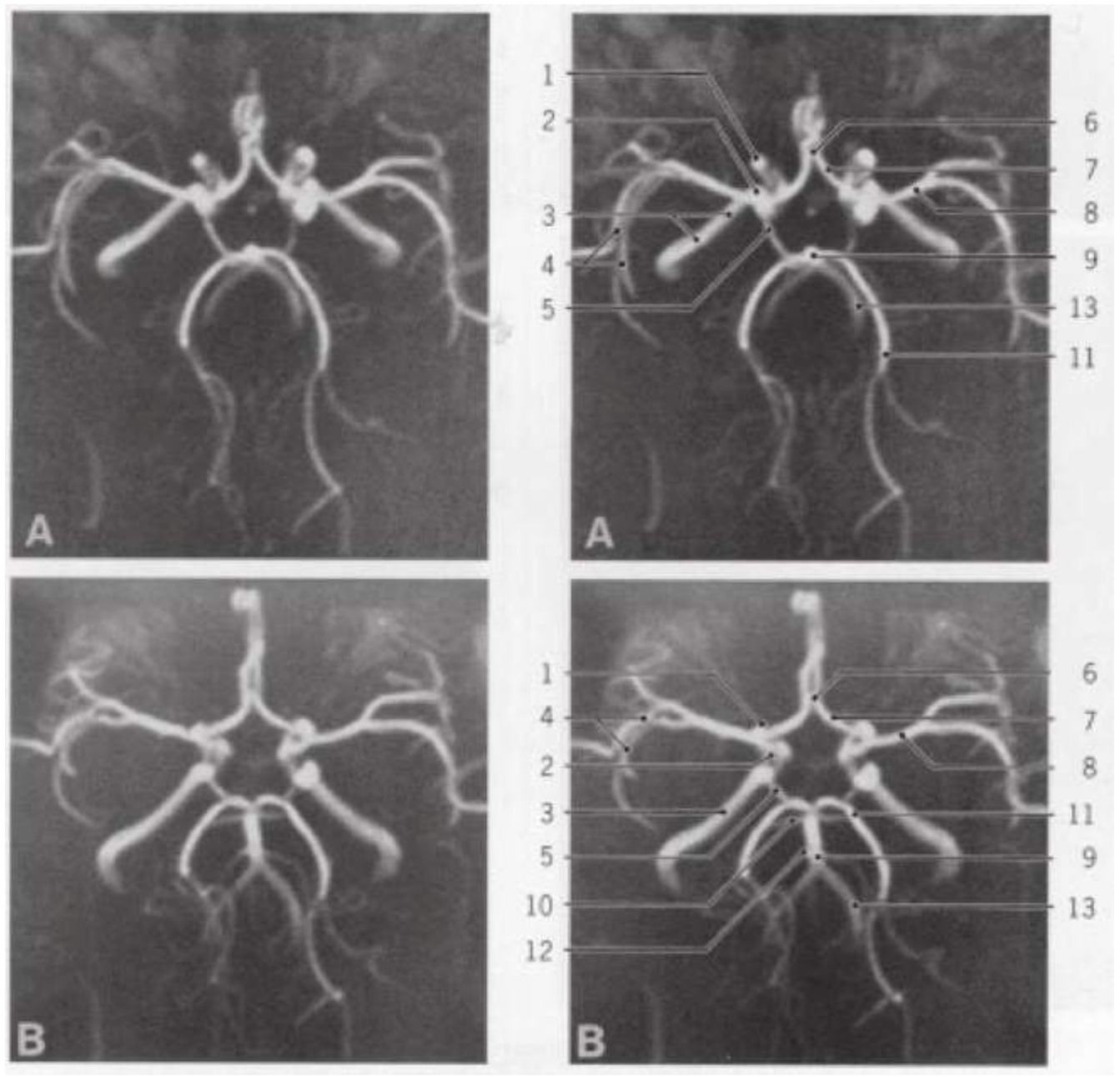
Posterior meningeal, anterior spinal, posterior inferior cerebellar, vertebrals fuse to form the basilar artery.

Basilar artery Branches

Anterior inferior cerebellar, pontine perforators, superior cerebellar, posterior cerebral.

posterior Cerebral artery Branches

Posterior thalamoperforators, medial posterior choroidal, lateral posterior choroidal, thalamogeniculates, inferior temporals, parieto-occipital, calcarine, posterior pericallosal.



Cerebral arteries, MR angiography, circle of Willis

1: Internal carotid artery, "siphon"
2: Internal carotid artery in cavernous sinus
3: Internal carotid artery in carotid canal
4: Insular branches of middle cerebral artery
5: Posterior communicating artery
6: Anterior communicating artery
7: Anterior cerebral artery
8: Middle cerebral artery
9: Basilar artery
10: Superior cerebellar artery
11: Posterior cerebral artery
12: Anterior inferior cerebral artery (AICA)
13: Vertebral artery

Figure 23-1. Mr angiography demonstrating circle of Willis. (Reproduced, with permission, from Fleckenstein P, Tranum-Jensen J. Anatomy and Diagnostic Imaging. 2nd ed. Philadelphia, PA: WB Saunders; 2001: 244.)

arterial

Circle of Willis is complete in only approximately one fifth of persons (see Figure 23-1).

Head Trauma

anatomy and physiology

scalp

- The scalp consists of five layers.
- Highly vascular structure, may be the source of major blood loss.

skull

- Rigid and inflexible (fixed volume).
- Composed of the cranial vault and base.

WARD TIP

Layers of the **sCALP:**
skin
Connective tissue
Aponeurosis (galea)
Loose areolar tissue
Pericranium

WARD TIP

CN III runs along the edge of the tentorium cerebelli.

Brain

- Makes up 80% of intracranial volume.
- Partially compartmentalized by the reflections of dura (falx cerebri and tentorium cerebelli).

Cerebrospinal Fluid (CSF)

- Formed primarily by the choroid plexus at a rate of ~500 cc/day with 150 cc of CSF circulating at a given moment.
- Cushions the brain.

Cerebral Blood Flow

- Brain receives ~15% of cardiac output.
- Brain responsible for ~20% of total body O_2 consumption.

Cerebral Perfusion Pressure (CPP)

- CPP = MAP - ICP.
- MAP = mean arterial blood pressure (avoid systolic blood pressure [SBP] < 90 mmHg).
- ICP = intracranial pressure (ICP > 20 mmHg should be treated).
- Maintaining CPP between 50 and 70 mmHg in nonoperative brain injury is the fundamental treatment.

WARD TIP

Concept of CPP is important in a hypertensive patient. Lowering the BP too fast will also decrease the CPP, creating a new problem.

Monro–Kellie Hypothesis

The sum of the volume of the brain, blood, and CSF within the skull must remain constant. Therefore, an increase in one of the above must be offset by decreased volume of the others.

Assessment

- History.
- Identify mechanism and time of injury, loss of consciousness, concurrent use of drugs or alcohol, medications that may affect pupillary size (e.g., glaucoma medications), past medical history (especially previous head trauma and stroke with their residual effects, and previous eye surgery, which can affect pupillary size and response), and the presence of a "lucid interval."

WARD TIP

Hypotension is usually not caused by isolated head injury. Look for other injuries in this setting.

Vital Signs

Cushing reflex:

- Brain's attempt to maintain the CPP.
- Hypertension and bradycardia in the setting of increased ICP.

Physical Exam

- Search for signs of external trauma such as lacerations, hemotympanum, ecchymoses, and avulsions, as these may be clues to underlying injuries such as depressed or open skull fractures.
- Anisocoria (unequal pupils) is found in a small percentage (10%) of normal people; however, anisocoria in the patient with head trauma is pathologic until proven otherwise.

WARD TIP

An enlarging pupil with a concurrent decrease in level of consciousness is strongly suggestive of uncal herniation.

Glasgow Coma Scale (GCS)

The GCS may be used as a tool for classifying head injury (see Figure 23-2):

Severe head injury	GCS 8 or less
Moderate head injury	GCS 9–13
Mild head injury	GCS 14 or 15

WARD TIP

Skin staples interfere with CT scanning and should therefore not be used until after CT scanning is complete.

Diagnostic Studies

- Assume C-spine injury in head injury patients and immobilize until cleared.
- Skull films have largely been replaced by computed tomography (CT) scan.

Eyes	Open spontaneously	4
	Open to verbal command	3
	Open to pain	2
	No response	1
Best motor response	Obeys verbal command	6
	Localizes pain to painful stimulus	5
	Flexion–withdrawal	4
	Decorticate rigidity	3
	Decerebrate rigidity	2
	No response	1
Best verbal response	Oriented and converses	5
	Disoriented and converses	4
	Inappropriate words	3
	Incomprehensible sounds	2
	No response	1
TOTAL		**15**

Figure 23-2. Glasgow Coma scale.

- Indications for head/brain CT:
  1. Neurologic deficit.
  2. Persisting depression or worsening of mental status.
  3. Depressed skull fracture or linear fracture overlying a dural venous sinus or meningeal artery groove (as demonstrated with skull x-rays).

Skull Fractures

linear (nondepressed)

stellate

- **Depressed:** Carries a much greater risk of underlying brain injury and complications, such as meningitis and posttraumatic seizures.
- **Basilar:** Signs include periorbital ecchymoses (raccoon's eyes), retroauricular ecchymoses (Battle's sign), otorrhea, rhinorrhea, hemotympanum, and cranial nerve palsies.

Diffuse Intracranial Lesions

Cerebral Concussion

- Transient loss of consciousness that occurs immediately following blunt, nonpenetrating head trauma, caused by impairment of the reticular activating system.
- Recovery is often complete; however, residual effects such as headache may last for some time.

diffuse axonal injury (dai)

- Caused by microscopic shearing of nerve fibers, scattered microscopic abnormalities.
- Frequently requires intubation, hyperventilation, CPP monitoring, and admission to a neurosurgical intensive care unit (ICU).
- Patients are often comatose for prolonged periods of time.
- Mortality is approximately 33%.

WARD TIP

Ring test for CSF rhinorrhea (in the presence of epistaxis): Sample of blood from nose placed on filter paper to test for presence of CSF. If present, a large transparent ring will be seen encircling a clot of blood.

WARD TIP

Typical scenario: A 20-year-old female has brief loss of consciousness following head injury. She presents to the ED awake but is amnestic for the event and keeps asking the same questions again and again. Think: Concussion.

WARD TIP

No mass lesion is seen on CT in DAI.

Focal Intracranial Lesions

Cerebral Contusion

- Occurs when the brain impacts the skull. May occur directly under the site of impact (coup) or on the contralateral side (contrecoup).
- Patients may have focal deficits; mental status ranges from confusion to coma.

intracerebral hemorrhage

Caused by traumatic tearing of intracerebral blood vessels. Difficult to differentiate from a contusion.

EXAM TIP

Typical scenario: A 19-year-old male with a head injury has loss of consciousness followed by a brief lucid interval. He presents to the emergency department (ED) in a coma, with an ipsilateral fixed and dilated pupil and contralateral hemiparesis. Think: Epidural hematoma.

epidural hematoma

- Collection of blood between the dura and the skull.
- Majority associated with tearing of the middle meningeal artery from an overlying temporal bone fracture.
- Typically biconvex or lenticular in shape (see Figure 23-3).
- Patients may have the classic "lucid interval," where they "talk and die." Requires early neurosurgical involvement and hematoma evacuation.
- Good outcome if promptly treated.

subdural hematoma

- Collection of blood below the dura and over the brain (see Figure 23-4). Results from tearing of the bridging veins, usually secondary to an acceleration–deceleration mechanism.

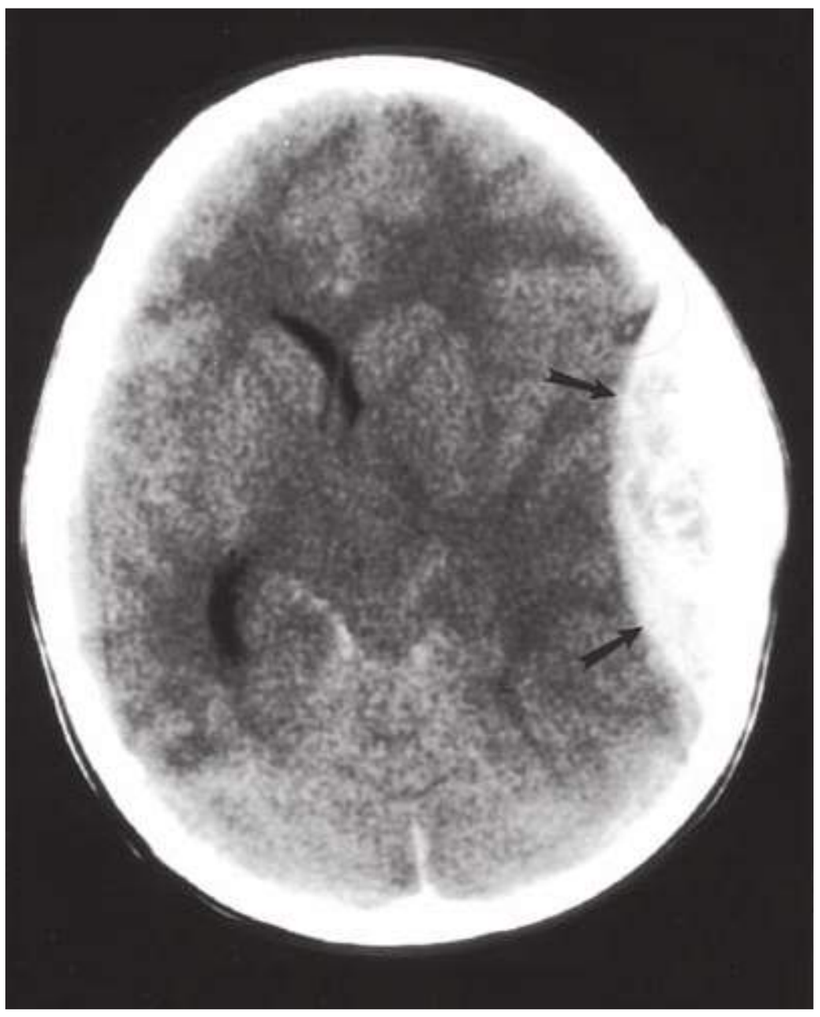


Figure 23-3. epidural hematoma. Arrows indicate the characteristic lens-shaped lesion. (Reproduced, with permission, from Schwartz SI, Spencer SC, Galloway AC, et al. Principles of Surgery. 7th ed. New York: McGraw-Hill; 1999: 1882.)



Figure 23-4. Subdural hematoma.

- Classified as acute (< 24 hours), subacute (24 hours–2 weeks), and chronic (> 2 weeks old).
- Acute and subacute subdurals require early neurosurgical involvement.
- Alcoholics and the elderly (patients likely to have brain atrophy) have increased susceptibility.

Management of Mild to Moderate Head Trauma

- Safe disposition of the patient depends on multiple factors.
- Any patient with a persisting or worsening decrease in mental status, focal deficits, severe mechanism of injury, penetrating trauma, open or depressed skull fracture, or seizures, or who is unreliable or cannot be safely observed at home, should be admitted for observation.
- Patients with mild and sometimes moderate head trauma, brief or no loss of consciousness, no focal deficits, an intact mental status, a normal CT scan, and reliable family members who can adequately observe the patient at home can often be discharged with proper discharge instructions.
- Discharge instructions should include signs and symptoms for family members to watch for, such as persisting or worsening headache, dizziness, vomiting, inequality of pupils, confusion.
- If any of the above signs are found, the patient should be brought to the ED immediately.

WARD TIP

Acute subdural hematomas have a high mortality—approximately one third to two thirds—mostly due to underlying brain contusion and shear.

WARD TIP

When in doubt, admit the patient for observation.

Management of Severe Head Injury (GCS < 9)

- Check airway, breathing, circulation (ABCs).
  - Patient needs intubation by definition.
  - Hyperventilation is not recommended, Pco_2 should be maintained at 35 mmHg and Pao_2 > 60 mmHg.
- ICP measurement via ventriculostomy should be done in all patients with severe traumatic brain injury and CT scan abnormalities.
- Maintain an adequate blood pressure (BP) with isotonic fluids. Avoid SBP < 90 mmHg.
  - Treatment of increased ICP: > 20 mmHg with mannitol.
  - Maintain CPP between 50 and 70 mmHg (preferably > 60 mmHg).
- Early feeding improves outcome.
- Use sequential compression device for deep venous thrombosis (DVT) prophylaxis.
- Corticosteroids are contraindicated in these patients.
- Consider prophylactic anticonvulsant therapy with phenytoin for 7 days.
- Hypothermia may be beneficial for neurological recovery.
- Treat the pathology whenever possible (e.g., surgical drainage of a hematoma).

EXAM TIP

Measures to lower ICP: **HIVe D**
Hyperventilation
Intubation with pretreatment and sedation
Ventriculostomy (burr hole)
elevate the head of the bed
Diuretics (mannitol)

hydrocephalus

Enlargement of the ventricles with excess CSF.

general

- Prevalence, –1%; congenital incidence, 1 in 1,000.
- Three general categories:
  - **Communicating:** Also known as normal-pressure hydrocephalus since flow remains between all the ventricles. Defective absorption by arachnoid granulations in the subarachnoid space.
  - **Noncommunicating (obstructive):** Increased pressure within the ventricular system due to disruption of CSF flow between the ventricles (e.g., aqueductal stenosis, tumor, cyst). May not affect all ventricles depending on the location of the block (e.g., aqueductal stenosis spares the fourth ventricle).
  - **Ex vacuo:** Atrophic parenchymal tissue loss resulting in dilated ventricles. Not pathologic hydrocephalus. (Example: Atrophy of caudate nuclei in Huntington's disease causes expansion of the lateral ventricles.)

Imaging

See Figure 23-5.

etiologies

- **Congenital:** Aqueduct stenosis, Dandy–Walker syndrome, Chiari malformation.
- **Acquired:**
  - Hemorrhage: Subarachnoid hemorrhages cause meningeal adhesions.
  - Infectious/inflammatory: Meningitis will cause meningeal adhesions.
  - Obstructing masses.
  - Postoperative (particularly in pediatric posterior fossa procedures).

EXAM TIP

Macewen's sign: Tapping on the head of a hydrocephalic infant produces a cracked pot sound.

Clinical Presentation

- **Communicating:** Classic triad of gait apraxia, dementia, and incontinence. Gait apraxia is usually first and can have a slow onset.

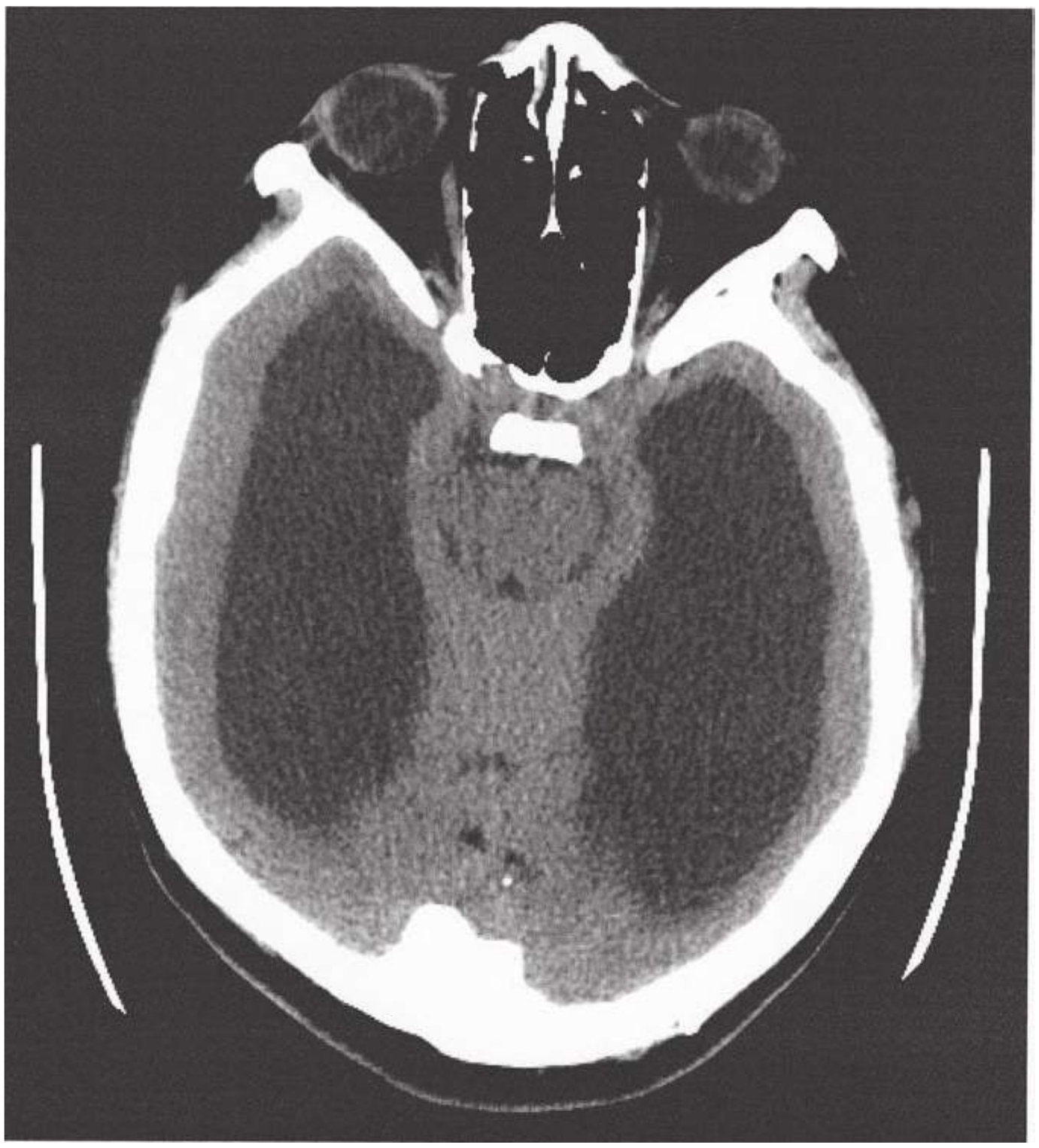


Figure 23-5. Brain CT demonstrating gross hydrocephalus. Note massive dilatation of lateral and third ventricles. The fourth ventricle is decompressed. There is a VP shunt tip in the body of the right lateral ventricle. Note deformity of calvarium, which is consistent with long-standing hydrocephalus.

- **Noncommunicating:** Headache, nausea/vomiting, ataxia, abducens palsy, Parinaud's syndrome.
- In children, check for bulging anterior fontanelle, increase in head circumference, irritability, poor feeding, and engorged scalp veins.

Treatment

- Acetazolamide to reduce CSF production and furosemide to promote diuresis (both only temporizing).
- Lumbar puncture: Used to quickly relieve CSF pressure. Considerable clinical improvement has high predictive value for success of shunt placement.
- Shunt placement:
  - Most commonly, a ventriculoperitoneal (VP) shunt is placed (see Figure 23-6). Alternatives include ventriculoatrial and ventriculopleural shunts.
- Shunts are placed similar to an extraventricular drain except that the catheter is subcutaneously tunneled behind the ear, where a valve is attached and placed in the subgaleal space. The catheter is then tunneled over the clavicle and to the destination outsource: peritoneum (VP, most common), atrium (ventriculoatrial), or pleura (ventriculopleural). Ventriculoatrial shunts occasionally cause pulmonary hypertension as a complication.

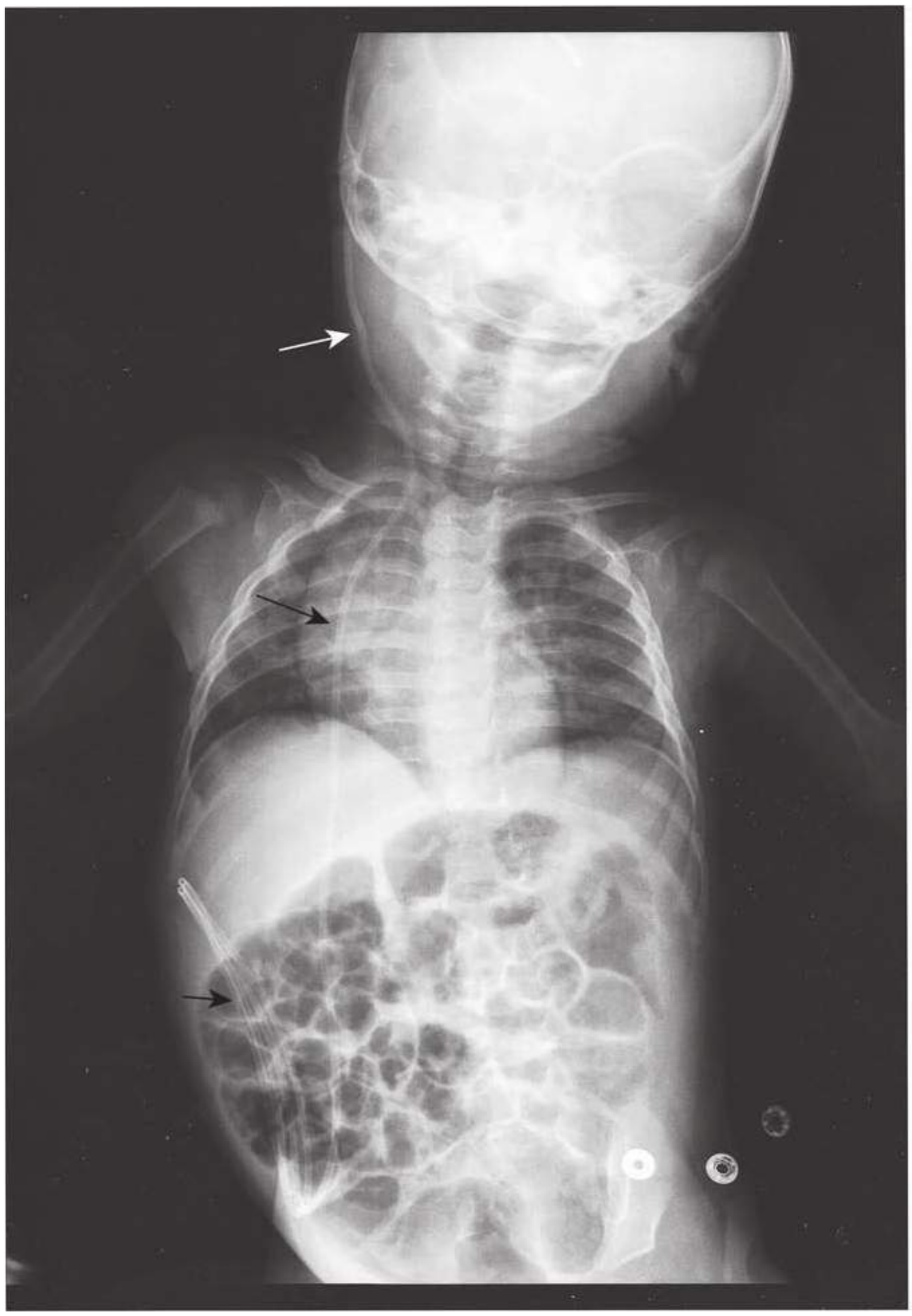


Figure 23-6. Plain radiograph ("shunt-o-gram") demonstrating VP shunt catheter coiled in the upper abdomen without evidence of kinks in this 8-month-old girl.

Complications

- Obstruction (usually proximal).
- Infection: Staphylococcus.
- Subdural hematoma.
- Patient growth—possible need for replacement of the distal catheter as the infant/child grows (shunt tip will pull out of peritoneal cavity).

CNS Tumors

general

- Most brain tumors present with progressive neurologic deficit, motor weakness, headache, or seizure.

- Tumor headache is usually due to elevated ICP.
- Any new-onset seizure in adulthood should prompt an aggressive search for a brain tumor.

presentation

- **Posterior fossa mass:** Headache, nausea/vomiting, ataxia, diplopia, Parinaud's syndrome, cranial nerve paresis, rotatory/vertical nystagmus.
- **Supratentorial mass:** Headache, nausea/vomiting, diplopia, Parinaud's syndrome, motor weakness, aphasia, tumor transient ischemic attack (TIA—hemorrhage/vascular compression).
- Dexamethasone may halt/reverse neurologic deterioration caused by vasogenic edema.

Primary Brain Tumors

glial tumor

CNS tumor arising from glial cells (astrocytes or oligodendrocytes).

astrocytoma

World Health Organization (WHO) grading system for astrocytomas most commonly used:

- Grade 1—Pilocytic astrocytoma: In children, consisting of cystic lesions, most often in the cerebellum, brain stem, or hypothalamus. Has good prognosis.
- Grade 2—Low-grade astrocytoma: Present at 20–40 years; median survival 5–10 years.
- Grade 3—Anaplastic astrocytoma.
- Grade 4—Glioblastoma multiforme: Present at 50–70 years; median survival 1–2 years; highly malignant with poor prognosis.

oligodendroglioma

- Male > female (3:2).
- Mostly occur in middle-aged adults, slow growing.
- Predilection for frontal and temporal lobes (40–70%).
- Uniform cells with round nuclei ("fried egg" appearance).
- May have mixed astrocyte component, here called oligoastrocytomas.
- Most frequently presents with a seizure.
- Surgery for symptomatic lesions, lesions > 5 cm.
- Responds well to chemo.

ependymoma

- Derived from cells lining the ventricles.
- Most occur in children.
- Fourth ventricle (most common).
- Spinal cord.
- Lateral ventricles.
- Most often there is hydrocephalus.
- Tends to disseminate through CSF ("seeding").
- Surgical resection.
- Radiation if in fourth ventricle or spinal cord.
- Chemotherapy is of little benefit.



WARD TIP

Preop imaging of the spinal neuraxis should be performed to detect CSF seeding.

meningioma

- Originates from arachnoid granulations.
- Most common benign brain tumor.
- Highest incidence in sixth to seventh decades.
- Seen at superior convexities, sphenoid wing, orbital rim, cerebellar tentorium, ventricles.
- Rarely invasive or metastatic; many discovered incidentally.
- Observe with annual CT scans if asymptomatic and < 2 cm.
- Surgical excision if symptomatic.
- External beam radiotherapy/gamma knife if subtotal resection or unresectable.

pituitary adenoma

- Associated with multiple endocrine neoplasia (MEN) syndrome.
- Seen at the sella turcica with parasellar extension.
- May envelop carotid arteries.
- Cell of origin may be chromophobe (null cell or prolactinoma), acidophil (growth hormone [GH], causing acromegaly), or basophil (adrenocorticotropic hormone [ACTH]—Cushing's syndrome).
- Generally slow, progressive enlargement.
- Headache, bitemporal hemianopsia (superior to inferior loss).
- **Prolactinomas (prolactin secreting):**
  - Medical treatment (dopamine agonists).
  - Surgical excision if no response to therapy, through transsphenoidal approach.
- **Acromegaly (growth hormone secreting):**
  - Surgical resection—50% cure.
  - Avoid surgery in asymptomatic elderly patients as there is no survival benefit.
  - Medical therapy with octreotide (somatostatin analogue).
- **Cushing's disease (cortisol secreting):**
  - Surgery is the treatment of choice—85% cure.
  - Nonfunctional adenomas.
  - Observe if asymptomatic; otherwise, surgical resection.

EXAM TIP

- Unilateral acoustic neuromas are common.
- Symptoms of hearing loss, vertigo, tinnitus.
- Bilateral indicative of NF2.

neuroma

- CN VIII affected most frequently (acoustic neuroma).
- Typical extension of acoustic neuroma into cerebellopontine angle with possible compression of CN V, VII, IX, or X.
- Usually unilateral.
- Bilateral CN VIII neuromas pathognomonic for neurofibromatosis type 2 (NF2).
- Unilateral progressive hearing loss (sensorineural), tinnitus, disequilibrium, possible vertigo.
- Perform pretreatment audiometric and vestibular testing.
- Surgical excision or stereotactic radiosurgery.
- Conventional radiotherapy (RT).

Craniopharyngioma

- Most occur in childhood.
- Anterior superior pituitary margin, may extend upwards toward third ventricle; may compress optic chiasm or pituitary gland.
- Benign but difficult to cure.

- Arises from remnants of the craniopharyngeal duct or Rathke's pouch.
- Headache and visual disturbance.
- Preop endocrinological evaluation.
- **Treatment:** May be observed; surgical resection if symptomatic.

hemangioBlastoma

- Tend to appear clinically in middle adulthood.
- Associated with Von Hippel–Lindau disease (dominant inheritance).
- Most common adult posterior fossa tumor.
- Spinal cord (70% have associated syringomyelia).
- Benign.
- Ataxia, dizziness, concurrent hepatic, renal, or pancreatic cysts.
- Surgery curative in sporadic cases.
- RT to slow growth if surgically unresectable.

glomus tumors

Tumors arising from paraganglion cells:

- **Glomus jugulare tumor:** Since located underneath floor of middle ear, typical presentation involves deafness, facial palsy, and possibly a palpable mass anterior to the mastoid eminence.
  - **Treatment:** Surgery with possible radical mastoidectomy followed with RT.
- **Carotid body tumor:** Painless mass below angle of jaw. May affect other cranial nerves in area. Small percentage of patients have experienced TIAs.
  - **Treatment:** Surgery; **no RT.** Possible embolization prior to surgery.

Metastatic Tumors

- More than 50% of brain tumors are metastatic in origin.
- Most disseminate hematogenously.
- Incidence of cerebral metastases is increasing.
- Common sources: Bronchogenic lung cancer (40%), breast cancer (19%), melanoma (10%), colon adenocarcinoma (7%).
- Most present with progressive focal neurologic deficit or signs/symptoms of increased ICP.
- Certain metastases are more likely to hemorrhage: Melanoma, renal cell carcinoma, choriocarcinoma.
- Most metastases occur in the cerebral hemispheres at the **gray–white junction** or in the cerebellum.

imaging

- CT or magnetic resonance imaging (MRI—preferred).
- Typically, metastases are well circumscribed.
- Usually, significant surrounding edema greater than that seen with primary brain tumors.
- Metastases usually enhance (completely or ring enhancement).

evaluation

Search for a primary source:

- CT chest/abdomen/pelvis.
- Bone scan, mammogram in women, guaiac for occult blood.

EXAM TIP

Von Hippel–Lindau Disease

- Autosomal dominant (chromosome 3).
- Multiorgan angiomatosis.
- CNS tumors are benign hemangioblastomas.
- Other organs: Spinal cord, eye, adrenal glands, kidneys, pancreas.
- Tendency for renal cell carcinoma and pheochromocytoma.

WARD TIP

A solitary cerebellar mass in an adult is presumed a metastasis until proven otherwise.

management

- Biopsy for diagnosis if no other source identified.
- Resect most solitary symptomatic lesions and treat with RT postoperatively.
- If multiple metastases, proceed directly to RT.

Spine

deFinitions

- **Spondylosis:** Degenerative changes in spine; arthritis.
- **Spondylolisthesis:** Subluxation of one vertebral body on another.
- **Spondylolysis:**
  - Fracture or defect in pars interarticularis.
  - Mostly congenital at L5 level (spina bifida occulta).
  - If due to congenital/degenerative etiology generally does not require surgical intervention.
  - Traumatic spondylolysis requires spinal fusion.

low BaCk pain

See Orthopedics chapter.

Spinal Trauma

general

- Spinal trauma may involve injury to the spinal column, spinal cord, or both.
- Over 50% of spinal injuries occur in the cervical spine (see Figure 23-7), with the remainder being divided between the thoracic spine, the thoracolumbar junction, and the lumbosacral region.
- As long as the spine is appropriately immobilized, evaluation for spinal injury may be deferred until the patient is stabilized.

anatomy

- There are seven cervical, 12 thoracic, five lumbar, five sacral, and four coccygeal vertebrae.
- The cervical spine is the region most vulnerable to injury.
- The thoracic spine is relatively protected due to limited mobility from support of the rib cage (T1–T10); however, the spinal canal through which the spinal cord traverses is relatively narrow in this region. Therefore, when injuries to this region do occur, they usually have devastating results.
- The thoracolumbar junction (T11–L1) is a fairly vulnerable region as it is the area between the relatively inf exible thoracic region and the f exible lumbar region.
- The lumbosacral region (L2 and below) contains the region of the spinal canal below which the spinal cord proper ends and the cauda equina begins.

pathology and pathophysiology

Spinal injuries can generally be classified based on:

- Fracture/dislocation type (mechanism, stable vs. unstable).
- Level of neurological (sensory and motor) and bony involvement.
- Severity (complete vs. incomplete spinal cord disability).

WARD TIP

Tuberous sclerosis clinical triad:

- Mental retardation
- Adenoma sebaceum (actually perivascular fibromata)
- Seizures

WARD TIP

Patients on a backboard for a prolonged period of time are at risk for the formation of pressure ulcers.

WARD TIP

Mechanisms suspicious for spinal injury:

- Diving.
- Fall from > 10 feet.
- Injury above level of shoulders (cervical spine).
- Electrocution.
- High-speed motor vehicle crash (MVC).
- Rugby or football injury (tackling).

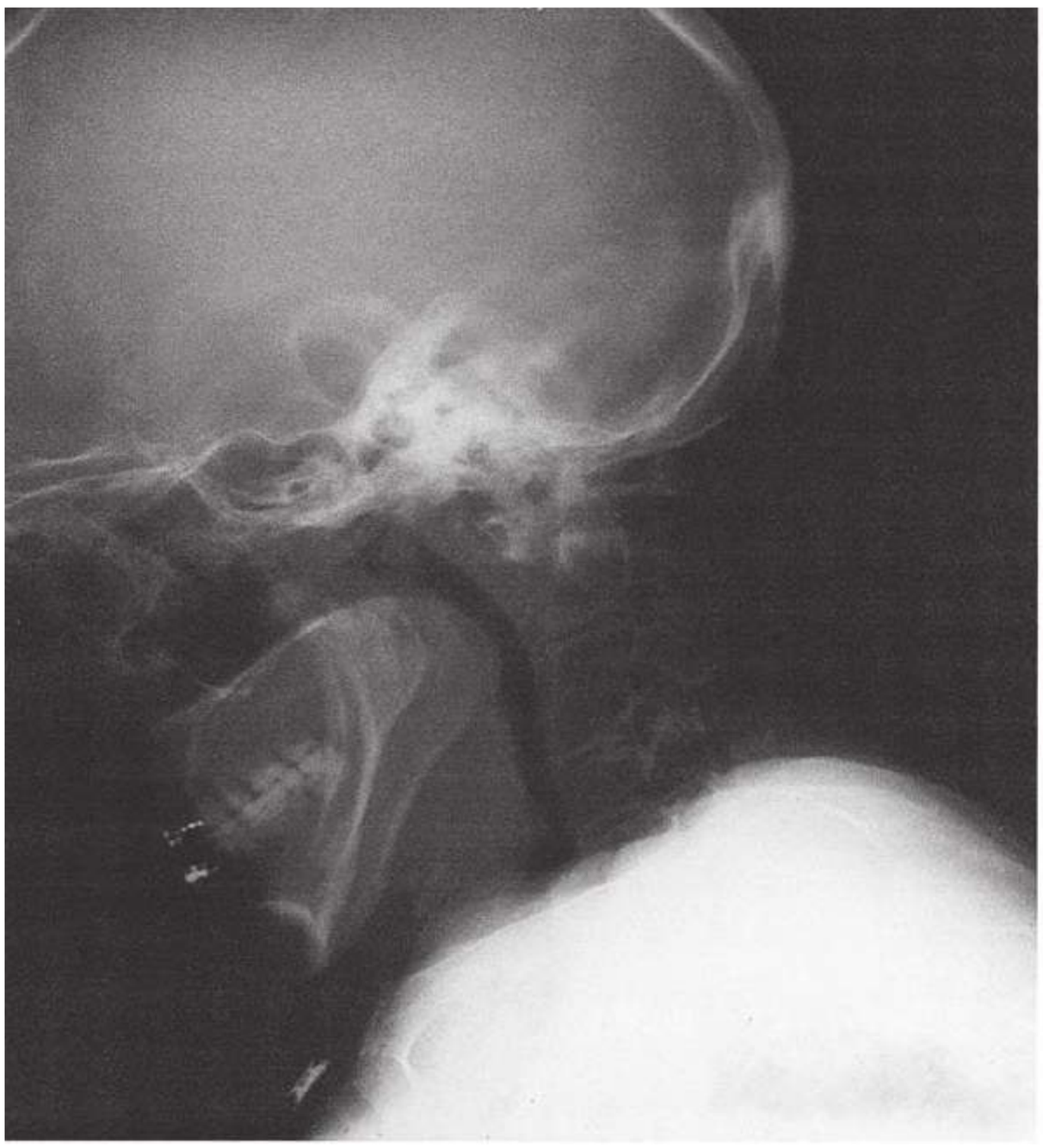


Figure 23-7. Atlantoaxial subluxation in a patient with rheumatoid arthritis.

neurogenic shock

- A state of vasomotor instability resulting from impairment of the descending sympathetic pathways in the spinal cord, or simply a loss of sympathetic tone.
- **Signs and symptoms:** Flaccid paralysis, hypotension, bradycardia, cutaneous vasodilation, and a normal to wide pulse pressure.

spinal shock

- State of flaccidity and loss of reflexes occurring immediately after spinal cord injury.
- Loss of visceral and peripheral autonomic control with uninhibited parasympathetic impulses.
- May last from seconds to weeks, and does not signify permanent spinal cord damage.
- Long-term prognosis cannot be postulated until spinal shock has resolved.

spinal cord injuries

Complete vs. incomplete:

- Complete spinal cord injuries demonstrate no preservation of neurologic function distal to the level of injury. Therefore, any sensorimotor function below the level of injury constitutes an incomplete injury.
- Sacral sparing refers to perianal sensation, voluntary anal sphincter contraction, or voluntary toe flexion, and is a sign of an incomplete spinal cord injury (i.e., better prognosis).

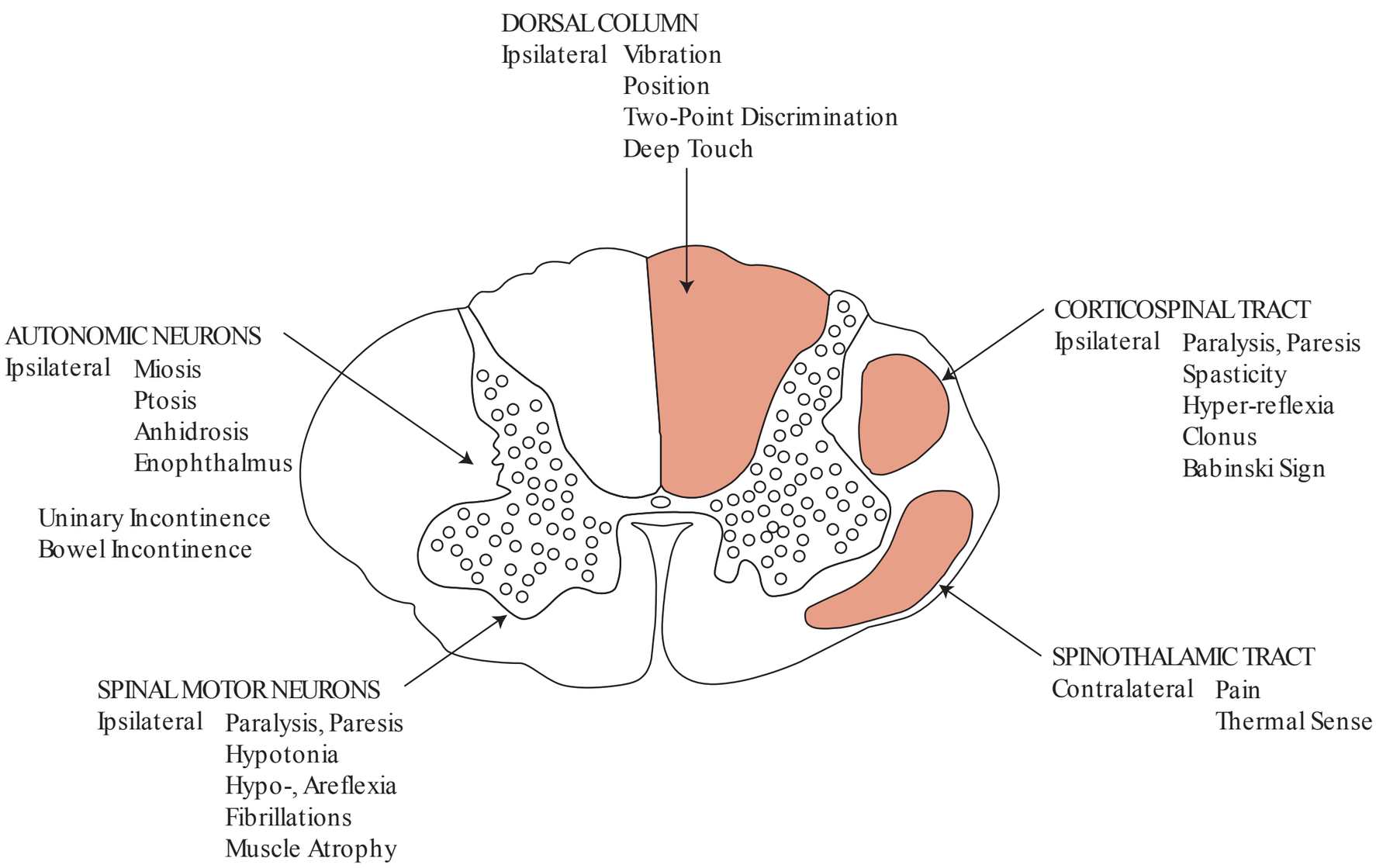


Figure 23-8. Cross-section of the spinal cord and tracts. (Reproduced, with permission, from Afifi AA, Bergman RA. Functional Neuroanatomy: Text and Atlas. New York: McGraw-Hill; 1998: 92.)

Physical exam

- Classification of spinal cord injuries as complete or incomplete requires a proper neurologic exam.
- The exam should include testing of the three readily assessable long spinal tracts (see Figure 23-8):
  - Corticospinal tract (CST):
    - Located in the posterolateral aspect of the spinal cord.
    - Responsible for ipsilateral motor function.
    - Tested via voluntary muscle contraction.
  - Spinothalamic tract (STT):
    - Located in the anteriolateral aspect of the spinal cord.
    - Responsible for contralateral pain and temperature sensation and tested as such.
  - Posterior columns:
    - Located in the posterior aspect of the spinal cord.
    - Responsible for ipsilateral position and vibratory sense and some light touch sensation.
    - Tested using a tuning fork and position sense of the fingers and toes.

WARD TIP

Deep tendon reflexes and sacral reflexes may be preserved in complete injuries.

Spinal Cord Syndromes

- **Anterior cord syndrome:**
  - Pattern seen with injury to the anterior portion of the spinal cord or with compression of the anterior spinal arteries (artery of Adamkiewicz).
  - Involves full or partial loss of bilateral pain and temperature sensation (STT) and paraplegia (CST) with preservation of posterior column function.
  - Often seen with flexion injuries.
  - Carries a poor prognosis.
- **Brown–Séquard syndrome:**
  - Pattern seen with hemisection of the spinal cord usually secondary to a penetrating injury, but may also be seen with disk protrusion, hematoma, or tumor.

- Consists of ipsilateral loss of motor function (CST) and posterior column function, with contralateral loss of pain and temperature sensation.
- **Central cord syndrome:**
  - Pattern seen with injury to the central area of the spinal cord often in patients with a preexisting narrowing of the spinal canal.
  - Usually seen with hyperextension injuries, its cause is usually attributed to buckling of the ligamentum flavum into the cord and/or an ischemic etiology in the distribution of branches of the anterior spinal artery.
  - Characterized by weakness greater in the upper extremities than the lower extremities, and distal worse than proximal.
  - Has a better prognosis than the other partial cord syndromes with a characteristic pattern of recovery (lower extremity recovery progressing upward to upper extremity recovery, then the hands recover strength).

EXAM TIP

Typical scenario: A 70-year-old male presents to the ED after a whiplash injury. He is ambulating well but has an extremely weak handshake. Think: Central cord syndrome.

Treatment of Spinal Cord Injuries

- Always start with the ABCs of trauma resuscitation.
- Maintain spinal immobilization throughout the resuscitation.
- Estimate level of neurologic dysfunction during the secondary survey.
- Obtain appropriate diagnostic studies.
- Establish early neurosurgical consultation.
- If blunt spinal cord injury is diagnosed, begin high-dose methylprednisone (must be given within 8 hours of injury and not for penetrating injury).
- Loading dose of 30 mg/kg over 15 minutes during hour 1, followed by a continuous infusion of 5.4 mg/kg/hr; the infusion is continued for 23 hours if the bolus is given within 3 hours of injury, or for 47 hours if the bolus is given within 8 hours of injury.

WARD TIP

Cervical spine films are indicated for:
- Tenderness along cervical spine.
- Neurologic deficit.
- Good mechanism of injury.
- Presence of distracting injury.
- Patients with altered sensorium.

Cervical Spine Fractures and Dislocations

General

As mentioned above, these injuries are usually classified on the basis of mechanism (flexion, extension, compression, rotation, or a combination of these), location, and/or stability.

Imaging

- Four views of the cervical spine are obtained (lateral, anteroposterior, oblique, and odontoid).
- A lateral view alone will miss 10% of cervical spine injuries.

Jefferson Fracture

- C1 (atlas) burst fracture.
- Most common C1 fracture.
- Consists of a fracture of both the anterior and posterior rings of C1.
- Results from axial loading such as when the patient falls directly on his or her head or something falls on the patient's head.
- Consider all C1 fractures unstable even though most are not associated with spinal cord injury (see Figure 23-9).

Odontoid Fractures

See Figure 23-10.

- Type 1: Involves only the tip of the dens (stable).
- Type 2: Involves only the base of the dens (unstable).
- Type 3: Fracture through the base and body of C2 (generally unstable).

WARD TIP

Some type 1 fractures may be stable when the transverse ligament remains intact.

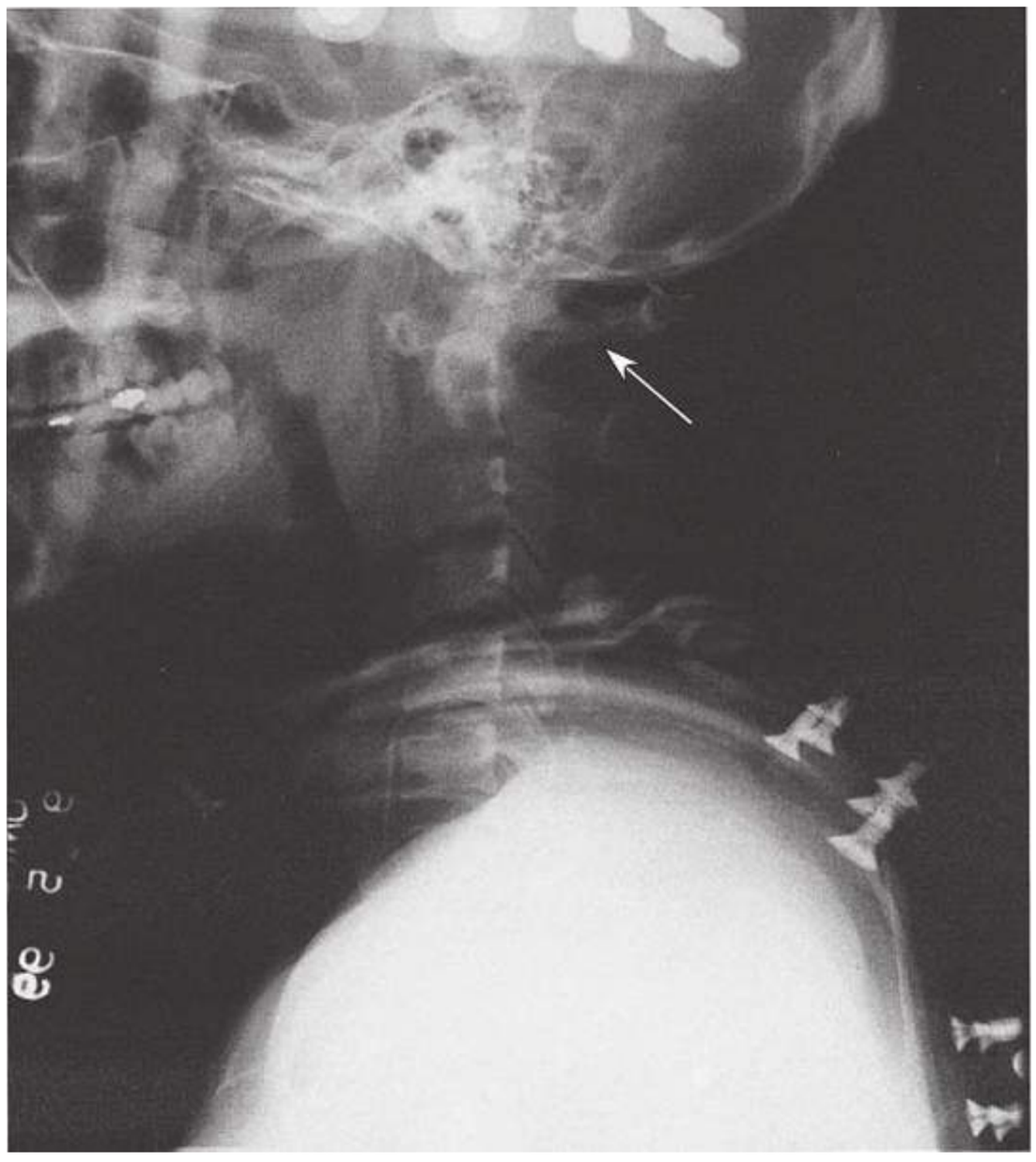


FIgure 23-9. Jef erson fracture.

Hangman's Fracture

See Figure 23-11.

- Fracture of both C2 pedicles ("posterior elements").
- Usually due to a hyperextension mechanism.
- Unstable fracture; however, often not associated with spinal cord injury because the spinal canal is at its widest through C2.

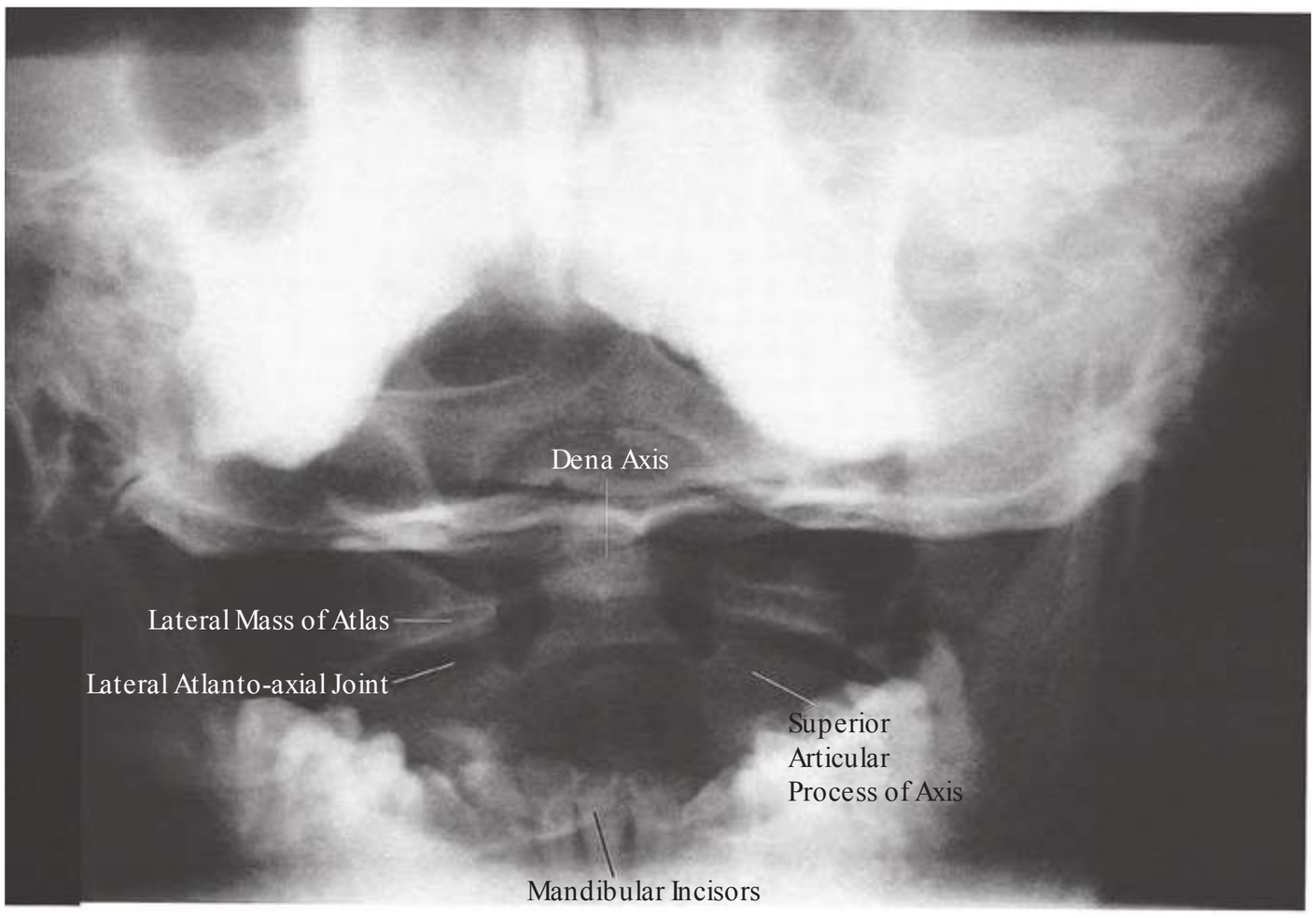


FIgure 23-10. Normal odontoid (open mouth) view of cervical spine.



Figure 23-11. Hangman's fracture.

Clay shoveler's Fracture

See Figure 23-12.

- Usually a flexion injury resulting in an avulsion of the tip of the spinous process (C7 > C6 > T1).
- May also result from a direct blow.

thoracic spine Fractures

- Most injuries occur at the junction between the relatively fixed upper thoracic spine and the mobile thoracolumbar region (T10–L5).
- The spinal canal in this region is narrow and the blood supply to this region of spinal cord is in a watershed area (the greater radicular artery of Adamkiewicz enters the spinal canal at L1, but provides blood flow as high as T4).
- Most thoracic spine fractures are caused by hyperflexion leading to a wedge or compression fracture of the vertebral body.
- Most fractures/dislocations in this area are considered stable because of the surrounding normal bony thorax; however, neurologic impairment resulting from injuries in this area is often complete.

WARD TIP

Frequency: L1 > L2 > T1

WARD TIP

May result in spinal cord compression.

thoracolumbar junction and lumbar spine Fractures and dislocations

- Results from axial loading and flexion.
- Potentially unstable.
- Neurologic injury is uncommon.

WARD TIP

Abdominal injuries frequently coexist with fracture-dislocations.



Figure 23-12. Clay shoveler's fracture.

Distraction or Seat-Belt Injury

- Frequently referred to as a **Chance fracture.**
- Horizontal fracture through the vertebral body, spinous processes, laminae, pedicles, and tearing of the posterior spinous ligament.
- Caused by an acceleration–deceleration injury of a mobile person moving forward into a fixed seat belt.

EXAM TIP

Median neuropathy due to carpal tunnel syndrome often involves the fingers but spares the palm, unlike compression at the pronator teres (palmar cutaneous branch of median nerve is outside the carpal tunnel).

Thoracic Outlet Syndrome (TOS)

- Subclavian artery/vein and brachial plexus pass through a space defined by the clavicle and first rib (thoracic outlet).
- Vascular compromise is more common than neurologic: Unilateral Raynaud's phenomenon, Adson's sign—loss of radial pulse on abduction and external rotation of the arm.
- Can present with T1 sensory loss, wasting of thenar muscles.

WARD TIP

Rotating head away from affected side with elevation of arm producing paresthesia/pain is suggestive of neurologic TOS. Concomitant reduction of radial pulse suggests vascular TOS.

Etiologies

- Fibrous band compressing C8/T1 roots (inferior trunk).
- Elongated C7 transverse process—"cervical rib."

Treatment

Surgical lysis of fibrous band or removal of C7 transverse process by either transaxillary or supraclavicular approach to thoracic outlet.

HIGH-YIELD FACTS IN

Cardiothoracic Surgery

Ischemic Heart Disease 382
- Definition 382
- Causes 382
- anatomy 382
- Pathophysiology 382
- Risk factors 382
- Signs and Symptoms 382
- Diagnosis 383
- treatment 383

Coronary Artery Bypass Grafting (CABG) 383
- Description 383
- indications 384
- Contraindications 384

PTCA versus CABG 384
- Preoperative Considerations 384
- Postoperative Considerations 385
- Complications 385
- Prognosis 385

Valvular Heart Disease 385
- mitral stenosis 385
- mitral insufficiency 387
- aortic stenosis 388
- aortic regurgitation 389
- tricuspid stenosis 391
- types of valve Prostheses 391

Lung Cancer 391
- types 391
- epidemiology 392
- etiology 392
- Signs and Symptoms 392
- Diagnosis 392
- treatment 392
- Diagnostic evaluation of a lung mass 393
- Solitary Pulmonary nodule 395
- Small Cell lung Cancer 395
- non-Small Cell lung Cancer 395

Thoracic Aortic Aneurysms 395
- Definition 395
- types 395
- anatomic Classification 395
- epidemiology 395
- Signs and Symptoms of expansion or Rupture 396
- Diagnosis 396
- treatment and Prognosis 397
- Complications 398

Thoracoabdominal Aneurysms 398
- Classification 398
- Diagnosis 398
- treatment 398
- Prognosis 399
- Complications 399

Ischemic Heart Disease

Definition

Myocardial injury caused by chronic or acute episodes of ischemia.

Causes

Disorders that affect coronary blood flow:

- Atherosclerotic coronary artery disease (most common).
- Valvular heart disease.
- Aortic dissection with or without involvement of coronary ostia.
- Vasculitis.
- Congenital coronary anomalies.
- Emboli, mainly tumor, calcium, vegetation.

anatomy

- **Right coronary dominance** (85–90% of patients): Right coronary artery (RCA) gives rise to posterior descending artery (PDA).
- **Left coronary dominance** (10–15%): Left circumflex artery gives rise to posterior descending artery. More common in males.
- **Codominance:** Occasionally, PDA arises from both the RCA and left coronary artery (LCA).
- Consider how a lesion from either side will affect the posterior and inferior walls.

PathoPhysiology

- Ischemic areas arise from lack of blood flow relative to the metabolic demands of the myocardium.
- The oxygen extraction already being high under normal metabolic conditions (75%), the heart must rely on increased blood flow to meet heightened demand.
- Determinants of demand: Wall tension (vis-à-vis preload, afterload, wall thickness), heart rate, level of contractility.
- An atherosclerotic plaque impedes flow significantly when coronary cross-sectional area is reduced by 75% (a 50% reduction in diameter).
- Coronary atherosclerotic lesions are usually multifocal and multivessel.
- Plaque rupture is the main cause of escalation of symptoms. Intermittent closure of dynamic plaques underlies symptoms of unstable angina.

EXAM TIP

Coronary atherosclerosis is the most common cause of cardiovascular morbidity and mortality in the Western world. Before age 70, men are more commonly affected than women by a ratio of 4:1. After age 70, it is 1:1.

Risk faCtoRs

- Hypertension (HTN)
- Smoking
- Hypercholesterolemia
- Abdominal obesity
- Diabetes
- Family history
- Psychosocial stress and depression

signs anDsymPtoms

- Fatigue
- Angina pectoris

- Dyspnea
- Edema
- Palpitations
- Syncope
- Abnormal heart sounds
- Seventy-five percent present with classic angina, 25% present atypically, many have "silent" myocardial infarctions (MIs) (particularly diabetics and elderly).

Diagnosis

- Electrocardiogram (ECG) may reveal ST segment elevations or depressions, inverted T waves, or Q waves.
- Stress test (to look at myocardial response when myocardial demand is increased).
- Echocardiography (localize dyskinetic wall segments, valvular dysfunction, estimate ejection fraction [EF]).
- Cardiac catheterization with angiography and left ventriculography (specifies coronary anatomy and sites of lesions to quantify the severity of the disease and vulnerable areas of myocardium, as well as to provide a road map for surgical or percutaneous intervention).
- Emerging technologies include: Computed tomography angiography and Coronary intravascular ultrasound.

Treatment

- Medical—aspirin, β-blockers, calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors, diuretics, nitrates.
- Percutaneous transluminal coronary angioplasty (PTCA).
- Surgical—coronary artery bypass grafting.

Coronary Artery Bypass Grafting (CABG)

Description

- Bypass of discrete areas of obstruction in coronary vessels using the internal mammary artery (IMA), radial artery, a reversed segment of greater saphenous vein, inferior epigastric artery, or gastroepiploic artery.
- IMA used in 95% of CABGs, usually to left anterior descending (LAD).
- Three or four grafts are used on average.
- Minimally invasive direct CABG (MIDCAB): Fewer incisions, no cardiopulmonary bypass or cardioplegia is used (performed on a beating heart, limited to single-vessel disease). Minimizes pain, recovery time, and chances of wound infection.
- Port access technique: Endovascular aortic occlusion and cardiopulmonary bypass with cardioplegia allows for broader use of MIDCAB (multivessel disease, combined valve-coronary artery surgery).
- Vessel acronyms:
  - LAD—left anterior descending
  - RCA—right coronary artery
  - LMCA—left main coronary artery
  - LCX—left circumflex artery
  - PDA—posterior descending artery
  - OMn—oblique marginal artery number 1, 2, 3, etc.
  - (L)(R)IMA—(left)(right) internal mammary artery

WARD TIP

Severity of heart failure graded by **Nyha Classification:**
Class I—no symptoms (fatigue, dyspnea, palpitations, angina)
Class II—symptoms with severe exertion
Class III—symptoms with mild exertion
Class IV—symptoms at rest

WARD TIP

Diffuse patterns of coronary vessel obstruction, as can occur in diabetes, may not be amenable to CABG.

Indications

- Mild angina or asymptomatic:
  - LMCA stenosis > 50%.
  - LMCA equivalent: Proximal LAD and LCX stenoses > 70%.
  - Triple-vessel disease (survival benefit > with abnormal left ventricular [LV] function: EF ≤ 0.50).
- Chronic stable angina:
  - Same as mild angina or asymptomatic.
  - Disabling angina despite maximal medical therapy (acceptable-risk patient).
  - Double-vessel disease with significant proximal LAD stenosis: Either EF < 0.50 or ischemia on noninvasive testing.
  - Single- or double-vessel disease without significant proximal LAD stenosis, but with a large area of viable myocardium and high-risk criteria on noninvasive testing.
- Unstable angina (UA)/non–ST elevation MI (NSTEMI):
  - LMCA stenosis ≥ 60%.
  - LMCA equivalent: Proximal LAD and LCX stenoses > 70%.
  - Ongoing ischemia unresponsive to maximal nonsurgical therapy.
- ST elevation MI (STEMI)/acute MI (AMI) (emergency or urgent CABG):
  - Failed angioplasty with persistent pain or hemodynamic instability.
  - Persistent or recurrent ischemia refractory to medical therapy.
  - Postinfarction ventricular septal rupture, mitral valve insufficiency or left ventricular aneurysm.
  - Cardiogenic shock in patients < 75 years with ST segment elevation or left bundle branch block (LBBB) or posterior MI who develop shock within 36 hours of MI.
  - Life-threatening ventricular arrhythmias in the presence of ≥ 50% left main stenosis and/or triple-vessel disease.
- Complications of PTCA stent replacement (rupture, dissection, thrombosis).

Contraindications

- Chronic congestive heart failure (CHF).
- Ischemic cardiomyopathy with no signs of angina or reversible ischemia.

PTCA versus CABG

PTCA is preferred over CABG when:

- Low-risk obstruction (single vessel, mild double vessel) is present, causing severe symptoms.
- Patient is at higher risk for complications from CABG.

Preoperative Considerations

- Evaluation for concurrent carotid disease (pre-CABG carotid endarterectomy may be required to reduce chances of perioperative stroke).
- Patencies of potential conduits are verified.
- Invasive monitoring (Swan–Ganz, central venous pressure [CVP], arterial line).
- Broad-spectrum antibiotics as needed.
- Aspirin discontinued 1–2 weeks preop.
- Warfarin discontinued 1 week preop.
- Antianginal meds continued until day of surgery.

WARD TIP

The IABP sits in the descending aorta (just distal to where the left subclavian takes off). It works by inflating during diastole and deflating during systole. Inflation increases coronary blood flow. Deflation creates a negative pressure gradient in the aorta, thereby reducing afterload.

- Intra-aortic balloon pump (IABP) for those who need it (augments cardiac output [CO]).

Postoperative Considerations

- **Short-term:**
  - Continued monitoring of cardiac parameters.
  - Chest tubes.
  - Atrial fibrillation prophylaxis.
  - Broad-spectrum antibiotics.
- **Long-term:**
  - Rehabilitation.
  - Sternum heals in 3–6 months.
  - Anticipate incisional pain.
  - Risk factor modification (hyperlipidemia, smoking, sedentary lifestyle).
  - Antiplatelet therapy (start the day after surgery).

WARD TIP

Operative mortality for CABG: 1–3% (elective), 5–10% (for acute MI).

Complications

- MI.
- Arrhythmias.
- Infection (particularly mediastinitis and sternal infection).
- Hemorrhage.
- Graft thrombosis.
- Sternal dehiscence.
- Tamponade.
- Postpericardiotomy syndrome.
- Stroke.

WARD TIP

Operative risk increases with (in descending order):

1. Cardiogenic shock
2. Need for inotropes
3. Preoperative IABP
4. Renal failure
5. Tricuspid disease

Prognosis

- Average survival rate at 1, 5, 10, and 15 years are 97%, 92%, 81%, and 66%, respectively
- Risk factors for mortality: Severe LV dysfunction, advanced age, emergent CABG for AMI or UA, female gender.
- Multivessel disease and left main disease demonstrate greater survival with surgical intervention over medical therapy.
- IMA has 98% 1-year and 90% 10-year patency.
- Saphenous vein has 80–90% 1 year and 60–70% 10-year patency.

Valvular Heart Disease

Mitral stenosis

Etiology

- Rheumatic heart disease (most common).
- Congenital (rare).

Epidemiology

More common in women.

Pathophysiology

- Mitral leaflets become thickened and calcified due to inflammation, resulting in commissural fusion in severe cases.

- Leads to pulmonary congestion and pulmonary hypertension, left atrial dilation, atrial fibrillation, reduced CO, right ventricular (RV) hypertrophy, and secondary tricuspid regurgitation.

Signs and Symptoms

- Dyspnea, DOE (dyspnea on exertion).
- Rales.
- Cough.
- Hemoptysis.
- Systemic embolism (secondary to stagnation of blood in enlarged left atrium).
- Loud S1, opening snap.
- Accentuated right ventricle precordial thrust.
- Signs of RV failure.
- Hoarse voice and dysphagia (secondary to enlarged left atrium impinging on recurrent laryngeal nerve and esophagus).

EXAM TIP

remember: Dilation of the left atrium is a major cause of atrial fibrillation.

diagnosis

- Murmur is mid-diastolic with opening snap, low-pitched rumble.
- Best heard over left sternal border between second and fourth interspace.
- Chest x-ray (CXR) may show straight left heart border secondary to enlarged left atrium and Kerley B lines from pulmonary effusion.
- ECG may show left atrial enlargement, RV hypertrophy, atrial fibrillation.
- Echocardiography demonstrates diseased valve, fish-mouth opening, and decreased cross-sectional area; 1.5–3.5 cm^2, mild stenosis; 1.0–1.5 cm^2, moderate stenosis; less than 1.0 cm^2, severe stenosis (normal mitral valve cross-sectional area 4–6 cm^2).
- Elevated transmitral pressure gradient > 10 mmHg (under normal conditions there is no transvalvular gradient).

EXAM TIP

Auscultatory triad of mitral stenosis:

- Increased first heart sound
- Opening snap
- Apical diastolic rumble

treatment

- **Medical:**
  - Asymptomatic patients need only endocarditis prophylaxis.
  - Symptomatic patients are treated with diuretics (to lower left atrial pressure), β-blocking agents, and/or calcium channel blocking agents (to maintain sinus rhythm).
  - Digoxin is helpful in controlling ventricular rate in patients who do go into atrial fibrillation.
  - Anticoagulation for atrial thrombus/fibrillation if present.
  - Percutaneous mitral valve balloon valvuloplasty if valve morphology allows.
- **Surgical:**
  - Indications for surgery:
    - NYHA Class III or IV symptoms.
    - Atrial fibrillation.
    - Worsening pulmonary hypertension.
    - Systemic embolization.
    - Infective endocarditis.
    - Class II patients > 40 of age.
  - Open commissurotomy:
    - Will suffice in 30–50% of cases.
    - Fused leaflets are incised, calcifications are debrided, problematic chordae are resected, papillary muscle heads may be split, and mitral ring added to prevent regurgitation (annuloplasty).
    - Valve replacement employed when excessive debridement would be required.

- Percutaneous balloon valvuloplasty not as effective in the long term, possibly because fused chordae cannot be corrected.
- Minimally invasive mitral valve surgery (port access technique) has recently been introduced.

Prognosis

- Ten years after commissurotomy, 7% require valve replacement.
- Yearly reoperation rates are 12% (balloon), 4% (commissurotomy), and 1.2% (valve replacement).
- Five-year survival after mitral valve replacement (MVR) 60–90%, 40–75% after 10 years (varies widely due to effect of risk factors, such as age, NYHA functional status, associated mitral insuff ciency, additional need for CABG).

mitRal insuffiCienCy

etiology

- Papillary muscle dysfunction from either ischemia or infarction (post-MI papillary muscle rupture causes massive regurgitation).
- Rupture of chordae tendineae (can happen spontaneously in otherwise healthy individuals).
- Valve destruction—scarring from rheumatic heart disease or destruction from endocarditis.
- Prolapse (billowing of one or both leaflets) frequently progresses to valvular incompetence.

Signs and Symptoms

- Dyspnea.
- Fatigue.
- Weakness.
- Cough.
- Atrial f brillation.
- Systemic emboli.
- Leads to pulmonary congestion, right-sided failure, left atrial dilation, atrial f brillation, and volume overload of left ventricle. Cardiac output increases, then decreases.

diagnosis

- Murmur is loud, holosystolic, high-pitched, apical radiating to the axilla.
- Wide splitting of S2 with inspiration (widening occurs in severe cases due to premature emptying of LV).
- S3 due to rapid f lling of LV by blood regurgitated during systole.
- ECG shows enlarged left atrium.
- Echocardiography demonstrates diseased/prolapsed valve and can be used to quantify mitral regurgitation (MR). The severity of the regurgitation is gauged as a function of the distance from the mitral annulus that the regurgitant jet can be visualized (e.g., into the pulmonary veins) and by the width of the regurgitant jet. The regurgitation is scored on a scale from 1 (mild) to 4 (severe).
- Leaflet thickness of 5 mm or more increases the likelihood of prolapse.

t reatment

- **Medical:**
  - Not def nitive but used until surgery or in poor surgical candidates.
  - Diuretics to reduce volume load, reduce LV diameter (and mitral annulus), and thus reduce regurgitant fraction.

EXAM TIP

Balloon valvuloplasty in mitral stenosis is an effective intervention, as it has a low incidence of restenosis, in contrast to aortic stenosis, and increases the mitral valve area to 2.0 cm^2.

WARD TIP

Carpentier's functional classification of mitral insuf ciency:

Type I—annular dilation or leaflet perforation with normal leaflet motion.

Type II—increased leaflet motion and prolapse.

Type III—restricted leaflet motion.

WARD TIP

Two parameters of LV function useful in decision making are EF and end-systolic diameter (ESD). Patients are referred for surgery when the left ventricular EF is < 60% or when the left ventricular ESD is > 45 mm.

- Cornerstone of medical management is afterload reduction by vasodilators (mainly ACE inhibitors), thus favoring aortic forward flow.
- Anticoagulation for atrial fibrillation.
- Mitral insufficiency has a good prognosis if LV function is preserved, and patient needs closed follow up with serial echocardiograms.

- **Surgical:**
  - Valve replacement or repair.
  - Indications for surgery: Symptoms despite medical management; severe MR with an identified structural abnormality, such as a ruptured chordae tendineae; development of pulmonary hypertension; or evidence of decline in left ventricular contractile function; atrial fibrillation; left atrium > 4.5–5 cm.
- **Mitral valve reconstruction:**
  - Involves resection of redundant areas of leaflets, chordal shortening, and ring annuloplasty (this corrects annular dilatation and stabilizes the repair).
  - Preferable to MVR in degenerative disease; MVR better in advanced deformity not amenable to reconstruction (e.g., due to rheumatic disease).

Prognosis

- Better late survival in nonrheumatic patients undergoing reconstruction vs. replacement.
- Opposite in rheumatic patients.

Aortic Stenosis

Etiology

WARD TIP

Congenital malformations account for about 50% of aortic valve operations.

- Calcific aortic stenosis (secondary to heavy dystrophic calcification of a congenitally abnormal valve).
- Degenerative aortic disease (idiopathic, older population).
- Congenital stenosis.
- Bicuspid aortic valve.
- Rheumatic heart disease.

Pathophysiology

Obstruction of flow leads to left ventricular hypertrophy (LVH) (concentric type) and decreased LV compliance, LV dilation, and left ventricular failure (LVF).

Signs and Symptoms

Usually asymptomatic early in course. Then:

- Dyspnea
- Angina and syncope—particularly during exercise. Peripheral resistance falls; LV pressure remains the same due to stenotic valve; CO cannot maintain blood pressure (BP), causing syncope; low BP to coronary arteries causes angina.
- Heart failure.
- HTN (consider associated aortic coarctation).
- Symptoms are associated with reduction in the aortic valve area from the normal 3–4 cm^2 to < 1 cm^2.

Diagnosis

- Forceful apex beat with normally located point of maximal impulse (PMI).
- Loud systolic ejection murmur, crescendo-decrescendo, medium pitched, loudest at second right interspace, radiates to carotids.

- S4 (presystolic gallop) frequently present secondary to reduced LV compliance.
- Paradoxical splitting of S2.
- Narrow pulse pressure.
- ECG may show LV strain pattern, LV hypertrophy, with or without inverted T waves.
- Echocardiography demonstrates diseased valve and quantifies severity; > 1.5 cm^2 mild stenosis, 1.0–1.5 cm^2 moderate stenosis, and < 1.0 cm^2 severe stenosis.
- Calcification of aortic valve may be seen on CXR.
- Patients > 40 years of age, consider coronary angiogram to rule out concurrent coronary artery disease.
- Hematologic abnormalities associated with severe aortic stenosis: Low platelet function and decreased levels of von Willebrand factor.

Treatment

- **Medical:**
  - Avoid strenuous activity.
  - Avoid afterload reduction.
- **Surgical:**
  - Indications for surgery:
    - Asymptomatic with high transvalvular gradient (> 50 mmHg) and LVH or declining EF.
    - Presence of symptoms: CHF is an indication of urgent intervention, while angina and syncope warrant elective surgical treatment.
    - Aortic valve area < 1.0 cm^2.
  - Aortic balloon valvuloplasty produces only temporary improvement as rate of restenosis is very high. Only potential role of valvuloplasty is in aged, frail, and possibly senile patients whose long-term survival is poor.
  - Valve replacement is definitive therapy. Nearly all patients attain symptomatic relief and improvement in EF while resolution of ventricular hypertrophy may require months.
  - Surgical mortality increases exponentially with decreasing LV function. Aortic valve replacement (AVR) in patients with CHF carries a mortality rate of up to 24%.
  - Intra-annular and supra-annular placement of prosthesis (latter for small annulus).
  - Ross procedure for AVR in patients with congenital aortic stenosis: Patient's own pulmonary valve is substituted (autograft), while a cryopreserved homograft (cadaveric) is used to replace the pulmonary valve. No need for anticoagulation, plus good durability (20-year failure rate of 15%).

Prognosis

Ten-year survival after AVR > 80% except in high-risk patients (e.g., severely impaired LV function, NYHA Class IV, pulmonary hypertension).

WARD TIP

Mean survival for patients with aortic stenosis and:
Angina: 5 years
Syncope: 2–3 years
Heart failure: 1–2 years

WARD TIP

LV strain pattern is ST segment depression and T wave inversion in I, AVL, and left precordial leads.

Aortic Regurgitation

Etiology

- Aortic root dilatation: Idiopathic (correlates with HTN and age), collagen vascular disease, Marfan's syndrome.
- Valvular disease: Rheumatic heart disease, endocarditis.

- Proximal aortic root dissection: Cystic medial necrosis (Marfan's syndrome again), syphilis, HTN, Ehlers–Danlos, Turner syndrome, third trimester.
- Aortitis: Rheumatoid arthritis, ankylosing spondylitis or Reiter disease.

Pathophysiology

Leads to LV dilation, eccentric hypertrophy, mitral insuffciency, cardiomegaly, CHF.

Signs and Symptoms

- Dyspnea, orthopnea, paroxysmal nocturnal dyspnea.
- Angina (secondary to reduced diastolic coronary blood flow due to elevated LV end-diastolic pressure).
- LVF.
- Wide pulse pressure.
- Bounding "Corrigan" pulse, "pistol shot" femorals, pulsus bisferiens (dicrotic pulse with two palpable waves in systole).
- **Duroziez's sign:** Presence of diastolic femoral bruit when femoral artery is compressed enough to hear a systolic bruit.
- **Hill's sign:** Systolic pressure in the legs > 20 mmHg higher than in the arms.
- **Quincke's sign:** Alternating blushing and blanching of the fngernails when gentle pressure is applied.
- **De Musset's sign:** Bobbing of head with heartbeat.

WARD TIP

Other conditions with wide pulse pressure:

- Hyperthyroidism
- Anemia
- Wet beriberi
- Hypertrophic subaortic stenosis
- HTN

diagnosis

- High-pitched, blowing, decrescendo diastolic murmur best heard over second right interspace or third left interspace, accentuated by leaning forward.
- Austin Flint murmur: Observed in severe regurgitation, low-pitched diastolic rumble secondary to regurgitated blood striking the anterior mitral leaflet (similar sound to MR).
- A2 accentuated (due to high pulse pressure in the aorta at the beginning of ventricular diastole).
- Hyperdynamic down and laterally displaced PMI secondary to LV enlargement.
- ECG shows LV hypertroph, high peaked T waves, and prominent Q waves.
- Echocardiography demonstrates regurgitant valve.

t reatment

- **Medical:**
  - Diuretics and afterload reduction: Afterload reduction achieved by ACE inhibitors. Nifedipine has been shown to delay the need for AVR.
  - Asymptomatic patients should have serial echocardiography to monitor for any systolic dysfunction or decreasing EF.
  - Endocarditis prophylaxis.
- **Surgical:**
  - Indications for surgery:
    - Asymptomatic with frst sign of declining LV function or rapid increase in cardiac size. AVR to be performed before end-systolic dimension exceeds 55 mm.
    - Presence of symptoms.
- Valve repair may be suitable for pure aortic insuffciency (no stenosis and no other valves involved) and in cases of aortic root aneurysm.
- Valve replacement is necessary for severe cases and is the only defnitive treatment.

Prognosis

- See Aortic Stenosis section.
- Valvular resection with annuloplasty: 10% reoperation rate at 2 years.

tRiCusPiD stenosis

etiology

Rheumatic heart disease, congenital, carcinoid.

Signs and Symptoms

- Peripheral edema
- Jugular venous distention (JVD)
- Hepatomegaly, ascites, jaundice

diagnosis

- Murmur is diastolic, rumbling, low pitched.
- Murmur accentuated with inspiration.
- Accentuated precordial thrust of right ventricle.
- Diastolic thrill at lower left sternal border.
- Best heard over left sternal border between fourth and fifth interspace.
- Echocardiography demonstrates diseased valve and quantifies transvalvular gradient.

treatment

- Valve replacement for most cases.
- Commissurotomy with annuloplasty used for commissural fusion.

WARD TIP

A rumbling diastolic murmur can be due to mitral stenosis or tricuspid stenosis. Tricuspid stenosis will increase with inspiration.

WARD TIP

Heart block is a common complication in tricuspid valve replacement due to the close proximity of the conduction bundle to the tricuspid annulus.

tyPes of valve PRostheses

Mechanical Prostheses

- St. Jude.
- Offer greater durability (15-year failure rate 5%), but need for lifelong anticoagulation (with associated risk of hemorrhagic complications).
- A better choice for the young (fewer re-operations).

Bioprostheses

- Porcine or bovine xenografts.
- Fewer thromboembolic concerns (usually no need for anticoagulation) but less durable (15-year failure rate 50%).
- Better choice for the elderly.
- Calcification can complicate use in the young.

Lung Cancer

tyPes

- **Small cell lung cancer:**
  - Represents 20% of all lung cancers and 80% of centrally located.
  - Neuroendocrine in origin.
  - Sensitive to chemotherapy (cisplatin and etoposide) and x-ray therapy (XRT).

- Usually nonresectable at time of diagnosis (< 5% candidates for surgery).
- Five-year survival: Very poor prognosis (2–4 months from diagnosis to death).
- Only T1, N0, M0 stage has 50% 5-year survival.

- **Non-small cell lung cancer:**
  - Includes squamous cell (SCCA), large cell, and adenocarcinoma (ACA).
  - ACA most common lung cancer (45% of all lung cancers), and 75% are peripherally located.
  - ACA metastasizes earlier than SCCA and more frequently to the central nervous system.
  - SCCA represents 30% of all lung cancers and two thirds are centrally located.
  - Large cell carcinoma accounts for 10% of all lung cancer, tends to occur peripherally, and metastasizes relatively early.
- Chemotherapy for non-small cell cancer (stage II or higher)—carboplatin, paclitaxel.
- Treated with surgery (debulking).
- Prognosis varies with stage.

EXAM TIP

SCCA has a rapid mitotic rate, and therefore is sensitive to chemotherapy. Surgery is not indicated.

epidemiology

- Leading cause of cancer death in both men and women in the United States.
- Cases have been decreasing in men but increasing in women.
- Smoking is by far the most important causative factor in the development of lung cancer.

EXAM TIP

Two types of cancer share a "s"entral location:

- Small cell
- Squamous cell

etiology

- Smoking
- Passive smoke exposure
- Radon gas exposure
- Asbestos
- Arsenic
- Nickel

EXAM TIP

Bronchoalveolar cancer, a type of adenocarcinoma, is not linked to smoking, and is more common in women.

signs and symptoms

- Cough.
- Hemoptysis.
- Stridor.
- Dyspnea.
- Hoarseness (recurrent laryngeal nerve paralysis).
- Postobstructive pneumonia.
- Dysphagia.
- Associated (paraneoplastic) syndromes (see Table 24-1).

EXAM TIP

Chronic cough is the most common symptom of lung cancer.

Diagnosis

See section on diagnostic evaluation of a lung mass below.

treatment

The two main types of lung cancer, small cell and non-small cell cancer, have different responses to radiotherapy, chemotherapy, and surgery (see Table 24-2).

TABLE 24-1. Syndromes associated with Lung Cancer

Horner syndrome	Sympathetic nerve paralysis produces enophthalmos, ptosis, miosis, ipsilateral anhidrosis
Pancoast's syndrome	Superior sulcus tumor injuring the eighth cervical nerve and the first and second thoracic nerves and ribs, causing shoulder pain radiating to arm
Superior vena cava syndrome	Tumor causing obstruction of the superior vena cava and subsequent venous return, producing facial swelling, dyspnea, cough, headaches, epistaxis, syncope. Symptoms worsened with bending forward, and on awakening in the morning
Syndrome of inappropriate antidiuretic hormone (SIADH)	Ectopic arginine vasopressin (AVP) release in the setting of plasma hyposmolality, producing hyponatremia without edema. Also caused by other lung diseases, CNS trauma or infection, and certain medications
Eaton–Lambert syndrome	Presynaptic nerve terminals attacked by antibodies, decreasing acetylcholine release, treated by plasmapheresis and immunosuppression; 40% associated with small cell lung cancer, 20% have other cancer, 40% have no cancer
Trousseau's syndrome	Venous thrombosis associated with metastatic cancer

TABLE 24-2. distinction between Small and Non-Small Cell Lung Cancer

Characteristic	Small Cell lung Cancer	non-Small Cell lung Cancer
Histology	Small dark nuclei, scant cytoplasm	Copious cytoplasm, pleomorphic nuclei
Ectopic peptide production	Gastrin, ACTH, AVP, calcitonin, ANF	PTH
Response to radiotherapy	80–90% will shrink	30–50% will shrink
Response to chemotherapy	Complete regression in 50%	Complete regression in 5%
Surgical resection	Not indicated	Stage I, II, IIIA
Included subtypes	Small cell only	Adenocarcinoma, squamous cell, large cell, bronchoalveolar
5-year survival rate—all stages	5%	11–83%

ACTH, adrenocorticotropic hormone; AVP, arginine vasopressin; ANF, atrial natriuretic factor; PTH, parathyroid hormone.

Diagnostic evaluation of a lung mass

Plain Film

- Most malignant nodules seen by 0.8–1 cm in diameter (may be seen smaller) (see Figure 24-1).
- Comparison with previous films whenever possible.
- Nodules stable for 2 years need no further evaluation.
- Plain film may be the only imaging modality necessary if there is obvious bony metastasis or bulky, contralateral mediastinal adenopathy.
- If abnormal nodule present, need computed tomography (CT).

EXAM TIP

Paraneoplastic Syndromes

Small cell carcinoma: ACTH, ADH.

Squamous cell carcinoma: PTH-related peptide.

ACTH secretion of small cell carcinoma is the most common paraneoplastic syndrome.

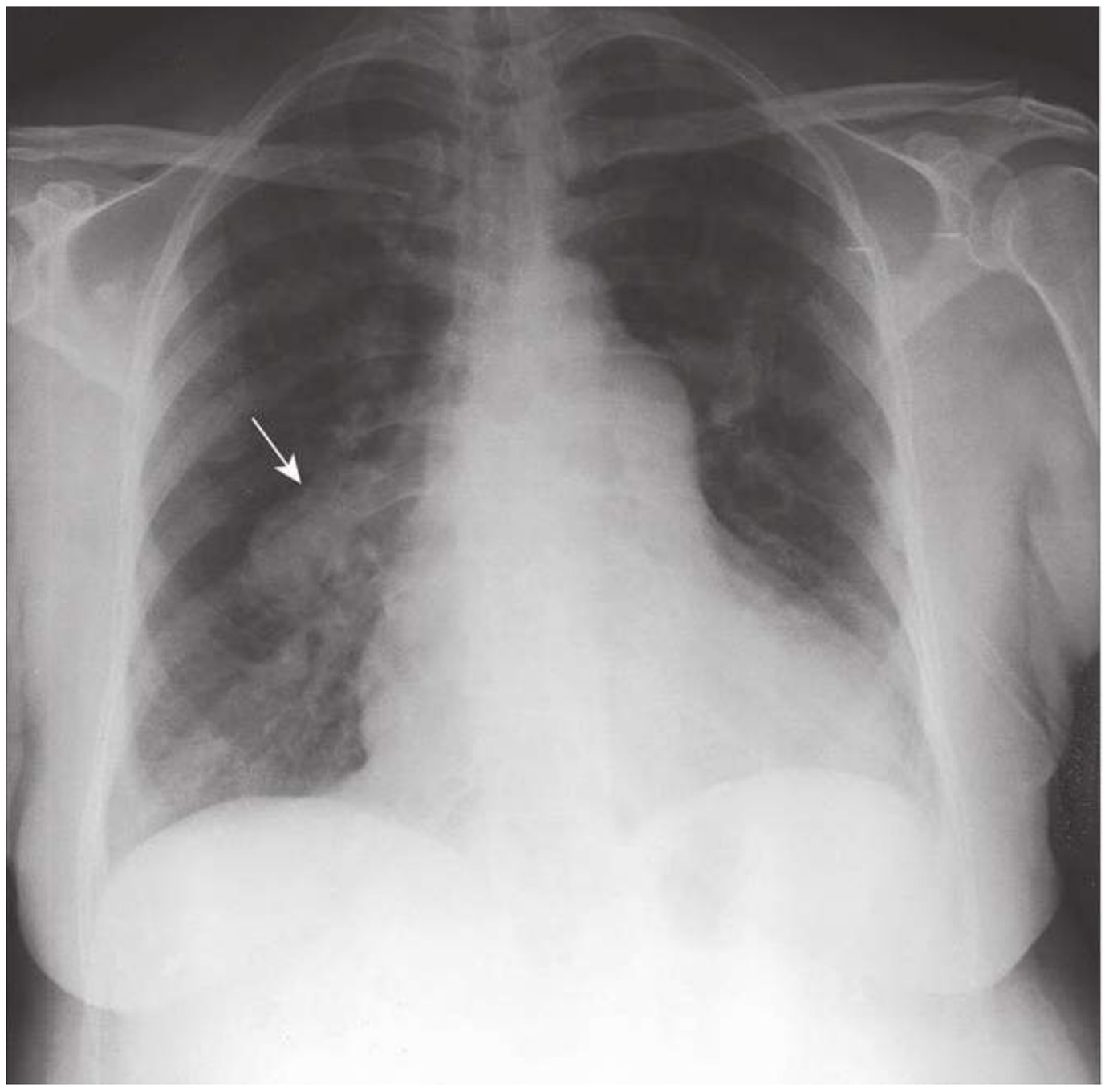


Figure 24-1. CXr demonstrating right middle lobe mass suspicious for malignancy.

Ct

- Provides better characterization and location of mass as well as detecting mediastinal invasion.
- Mediastinal lymph nodes can also be assessed, especially if greater than 1 cm.
- Should be extended to include liver and adrenal glands as these are frequent sites of metastasis.

Bronchoscopy

- Method of choice of centrally located masses (squamous cell and small cell).
- Specimens can be obtained via direct biopsy of visualized lesions, brushings, washings, or transbronchial needle aspiration (TBNA).
- The most important application of TBNA is staging of mediastinal lymph nodes.
- Risks of this procedure are respiratory arrest, pneumothorax, and bleeding.
- Although this method works well for centrally located lesions, it is poor when it comes to peripheral lung nodules.

transthoracic Needle Biopsy (tNB)

- Method of choice for peripherally located nodules. Most are CT guided.
- Sensitivity: 70–100%.

thoracentesis

Test of choice for patients with pleural effusion and suspected malignancy.

Solitary Pulmonary Nodule

- A single small (< 3 cm) intraparenchymal opacity that is reasonably well marginated.
- Most will be benign.
- Benign: Granulomas, hamartomas, or intrapulmonary lymph nodes.
- Malignant: Bronchogenic carcinoma.

Small Cell Lung Cancer

- Seventy percent metastatic at the time of diagnosis.
- Generally considered inoperable for cure.
- Two stages: Limited and extensive.
  - Limited: Confined to a single radiation portal.
  - Extensive: All others.

Non-Small Cell Lung Cancer

- Need to determine resectability: Chest CT and search for distant metastases.
- Malignant pleural effusion precludes curative resection.
- Tumor-node-metastasis (TNM) staging system.

Thoracic Aortic Aneurysms

Definition

- **Aneurysm:** Ballooning defect in the vessel wall.
- **Dissection:** Tear of the arterial intima.

Types

- **Degenerative:**
  - Due to abnormal collagen metabolism.
  - Seen with Marfan's and Ehlers–Danlos syndromes (usually ascending aortic aneurysms).
- **Atherosclerotic** (usually descending aortic aneurysms): Due to remodeling and dilatation of the aortic wall.

Anatomic Classification

- DeBakey type I: Ascending and descending aorta.
- DeBakey type II: Ascending aorta only.
- DeBakey type III: Descending aorta only.
- Stanford A: Ascending aorta (same as DeBakey I/II).
- Stanford B: Descending aorta (same as DeBakey III).
- Ascending aorta and aortic arch aneurysms are worse than descending aortic aneurysms.
- Expansion rate is –0.56 cm/year for arch aneurysms and –0.42 cm/year for descending aorta.

Epidemiology

- Six per 100,000 a year.
- Male-to-female ratio is 2:1.
- Familial clustering.
- Patients tend to be younger than those with abdominal aortic aneurysm (AAA).

Signs and Symptoms of Expansion or Rupture

- "Tearing" or "ripping" chest pain radiating to the back.
- Acute neurologic symptoms (syncope, coma, convulsions, hemiplegia).
- Palpable thrust may be seen in right second or third intercostal space.
- Pulsating sternoclavicular joint may be seen (secondary to swelling at the base of the aorta).
- Hoarseness.
- Stridor.
- Dysphagia.
- New aortic regurgitation murmur.
- Hemoptysis or hematemesis.
- Absent or diminished pulses.

Diagnosis

- **CXR** (Figure 24-2):
  - Widened mediastinum.
  - Abnormal aortic contour.
  - "Calcium sign": Reflects separation of intimal calcification from the adventitial surface.
- **Contrast CT** (Figure 24-3):
  - Sensitivity 85–100%.
  - Specificity 100%.
- **Magnetic resonance imaging (MRI):**
  - Excellent sensitivity and specificity.
  - Gives information about branch vessels that CT does not.
  - No need for contrast.
  - Limited to stable patients.
- **Angiography** (Figure 24-4):
  - Requires contrast dye like CT.
  - Invasive.

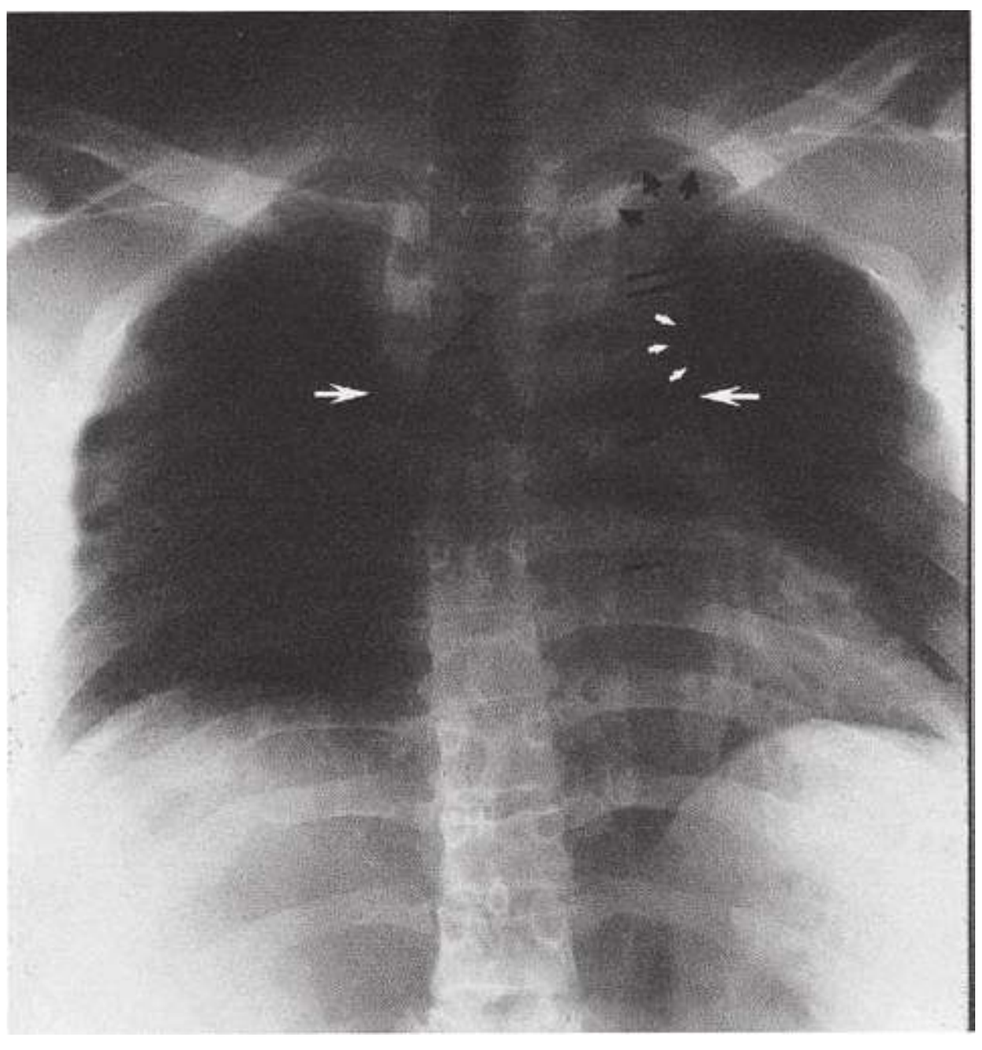


Figure 24-2. Thoracic aneurysm diagnosed on CXr.

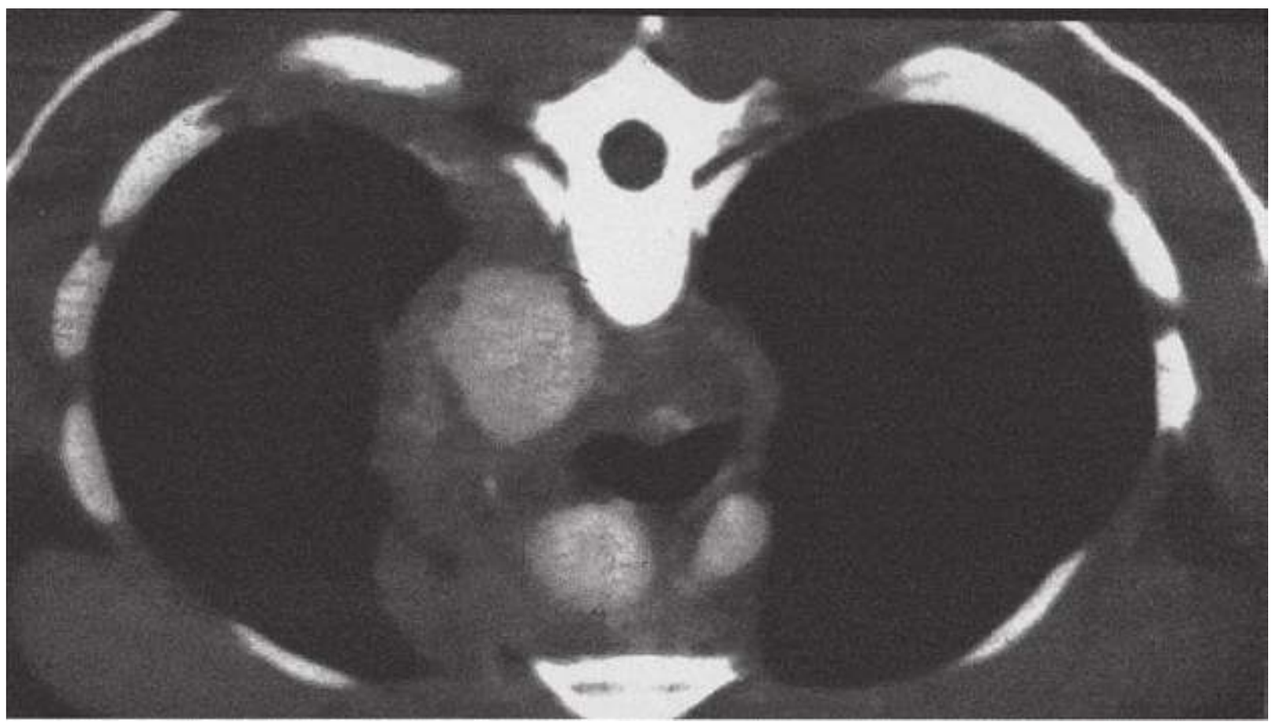


Figure 24-3. Thoracic aneurysm on CT.

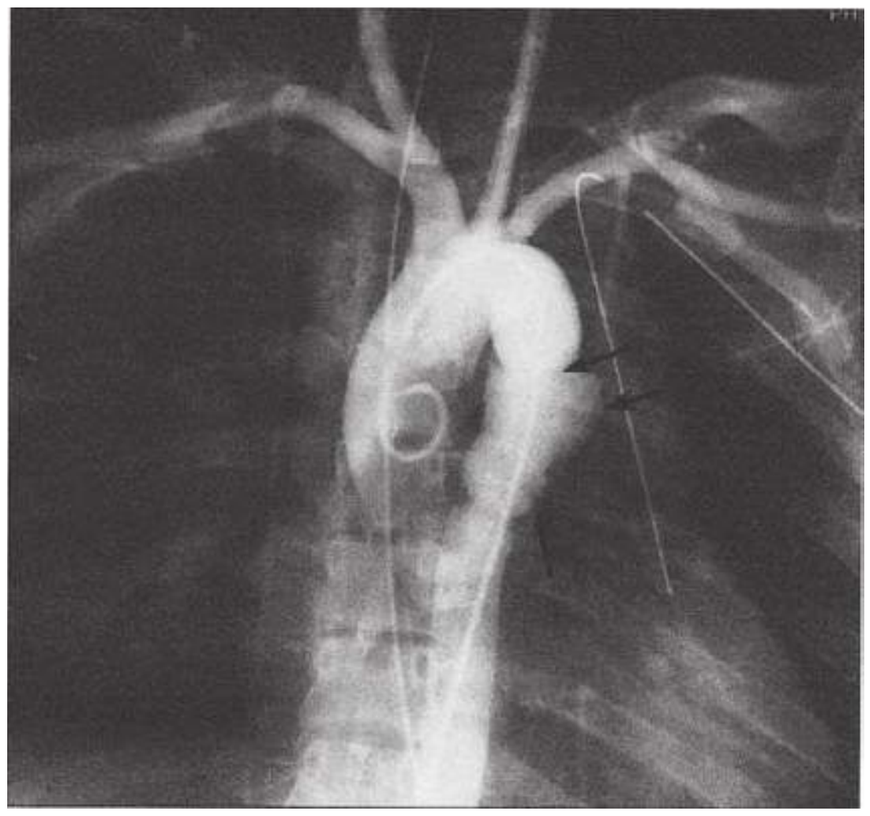


Figure 24-4. Angiogram demonstrating thoracic aneurysm.

- **Transesophageal echocardiography (TEE):**
  - Presence of intimal flap separating the true from the false lumen.
  - Features of the false lumen: Larger in diameter, slower blood flow velocity.
  - Can be used in relatively unstable patients as well.
- **Transthoracic echocardiography (TTE):**
  - Available at bedside.
  - Noninvasive.
  - Suitable for unstable patients.
  - Requires operator expertise.
  - Moderate ability to detect ascending and arch dissections; poor for detecting descending arch dissection.

Treatment and Prognosis

- **Medical:**
  - Control HTN with nitroprusside or labetalol.
  - Parenteral analgesia.
- **Surgical:**
  - For ruptured aneurysms, it is the only definitive therapy. Carries very high risk of mortality. Most patients die before reaching the operating room. Of those that reach the OR, < 50% survive.

EXAM TIP

Major complication of any thoracic aneurysm repair is paraplegia. Key is to avoid perioperative hypotension.

- Surgical repair is considered for acutely symptomatic, ascending aortic and aortic arch aneurysms > 5.5 cm, rapid increase in size (growth rate > 0.5 cm/year). Mortality rate is 10–15%.
- For degenerative aneurysms, the entire aortic root must be replaced.
- Atherosclerotic aneurysms can be repaired either via open approach (median sternotomy approach for ascending arch and posterolateral thoracotomy for descending arch) or via endovascular technique.
- Ascending and descending arch aneurysms are repaired with patient under cardiopulmonary bypass, anticoagulation, and in mild-moderate hypothermia.
- Aortic arch aneurysms are repaired with patient in circulatory arrest and profound hypothermia.

Complications

- Hemorrhage
- Paraplegia
- Stroke
- MI
- Visceral ischemia
- Death

Thoracoabdominal Aneurysms

Classification

Crawford classification:

- Type I: Most of descending thoracic aorta and abdominal aorta proximal to renal arteries.
- Type II: Most of descending thoracic aorta and abdominal aorta distal to renal arteries.
- Type III: Distal one half of descending thoracic aorta and abdominal aorta proximal to renal arteries.
- Type IV: Distal one half of descending thoracic aorta and abdominal aorta distal to renal arteries.

Diagnosis

Made incidentally (routine physical exam or imaging for other reasons) or on postmortem exam (for ruptured ones).

Treatment

- Elective repair undertaken after weighing risk vs. benefit.
- Open surgical approach decreasing in practice:
  - Type I: Thoracic incision.
  - Types II and III: Incision from sixth intercostal space into abdomen.
  - Type IV: Retroperitoneal incision from left flank to umbilicus.
- Endovascular repair of aortic aneurysm (EVAR):
  - Widespread use in the last decade except in urgent/emergent cases.
  - Need to have good preoperative imaging to evaluate anatomy of the aneurysm
  - Suitable landing zones proximal and distal to the graft: 1.5–2.0 cm, no major arterial branches near the neck of aneurysm.

- If a major artery (left subclavian, celiac axis, superior mesenteric artery, renal artery) is covered by the endovascular graft, then need a hybrid operation to bypass the covered artery.
- Complications:
  - Endoleak (leak through or around the graft) results in persistent enlargement of the aneurysm sac
  - Aortic dissection
  - Aneurysm rupture
  - Stent collapse
  - Stent migration
  - Injury to femoral and external iliac arteries while graft insertion

PRognosis

Overall mortality < 10%.

ComPliCations

- Risk of paraplegia 5–10%.
- Same as for thoracic aneurysm repair.

HIGH-YIELD FACTS IN

Transplant

Definitions 402

Brain Death 402
- Adult Criteria 402
- Non-Heart-Beating donors 402
- donor MANAgeMeNt 402

Transplant Immunology 402
- BAsiCs 402
- MHC i 402
- MHC ii 403
- MHC iii 403
- tissue typiNg 403
- CrossMAtCH 403

Immunosuppression 403
- Steroids 403
- CyClosporiNe (CsA) 404
- MyCopHeNolAte Mofetil (MMf) 404
- AzAtHiopriNe 404
- tACroliMus (fK-506) 404
- ANtitHyMoCyte gloBuliN (Atg) 404
- oKt 3 404

Rejection 405
- HyperACute 405
- ACute 405
- CHroNiC 405
- risK of MAligNANCy 405

Organ Preservation 406
- MAxiMuM tiMes for eACH orgAN 406

Kidney Transplantation 406
- BACKgrouNd 406
- CAuses of eNd-StAge reNAl diseAse (esrd) 406
- types of doNors 406
- AppropriAte reCipieNt 406
- CoMpliCAtioNs SpeCifiC to KidNey trANsplANtAtioN 407

Pancreas Transplantation 408
- iNdiCAtioNs 408
- exClusioNs for reCipieNts 408
- tiMiNg of operAtioN 408

Drainage Options 408
- CoMpliCAtioNs (SpeCifiC to pANCreAtiC trANsplANt) 409

Liver Transplantation 409
- iNdiCAtioNs 409
- types of liver trANsplANtAtioN 409
- evAluAtioN 409
- CoMpliCAtioNs 410

Small Bowel Transplantation 410
- iNdiCAtioNs 410
- operAtioN 410
- CoMpliCAtioNs 410

Cardiac Transplantation 410
- iNdiCAtioNs 410
- CoNtrAiNdiCAtioNs 411
- evAluAtioN 411
- CoMpliCAtioNs 411

Lung Transplantation 411
- types of operAtioNs ANd iNdiCAtioNs 411
- CoNtrAiNdiCAtioNs 412
- evAluAtioN 412
- CoMpliCAtioNs 412

Definitions

Autograft: Donor and recipient are same individual.
Allograft: Donor and recipient belong to same species but have different genetic makeup.
Isograft: Donor and recipient are genetically identical, for example, identical twin.
Xenograft: Donor and recipient are of different species.
Orthotropic: Transplant graft placed into its anatomic position.
Heterotopic: Transplant graft placed at different site.

Brain Death

Brain death is defined as irreversible loss of all functions of the brain including brainstem.

Adult Criteria

1. No cerebral function: Patient is in a deep coma, unresponsive to stimuli.
2. Absence of brainstem reflexes: No evidence of cranial nerve function; lack of reflexes (pupillary, corneal, cold water calorics, doll's eyes, pharyngeal and tracheal).
3. No spontaneous breathing: Apnea test.
   - **Requirements:** Normothermia, no central nervous system (CNS) depressants or neuromuscular blockers in effect. No severe electrolyte/acid base or endocrine disturbances.
   - **Optional tests:** Electroencephalogram, angiography, nuclear scan transcranial Doppler.

Non-Heart-Beating donors

Applies to patients with cardiac arrest near hospital, and to patients requesting removal of life support or in whom death is expected.

WARD TIP

If the patient does not qualify for organ donation, cornea and heart valves may still be used.

donor MANAgeMeNt

Goal: To optimize organ function avoid hypoxia, hypotension, and fever.

Transplant Immunology

BAsiCs

Major histocompatibility complexes (MHCs) present peptide antigens to T cells and are the major target of activated lymphocytes.

MHCi

- Found on all nucleated cells except cells in retina and brain. High level on antigen presenting cells.

- Consists of MHC coded α chain and non-MHC coded β chain, β2 microglobulin.
- Presents cytosolic proteins and viruses.
- Primary target for CD8 T cells in graft rejection.
- Gene loci: A, B, C.

MHC ii

- Constitutively expressed on Antigen presenting cells like B-lymphocytes, dendritic cells, macrophages, and other phagocytic cells, also can be induced on other cells by cytokines.
- Composed of alpha and beta chains.
- Binds peptides from extracellular proteins.
- Primary target for T-helper cells.
- Gene loci: DR, DQ, DP.

MHC iii

Class 3 genes encode complement proteins.

tissue typiNg

- Determination of MHC alleles in an individual to minimize differences in histocompatibility.
- Of above alleles, only A, B, and DR are used in routine tissue typing.
- Of these three genes, DR is most important to match, followed by B and then A, as evidenced by United Network for Organ Sharing (UNOS) data regarding graft survival.

Cr oss MAt CH

- Test for preformed cytotoxic antibody in serum of potential recipient.
- Donor lymphocytes are cultured with recipient serum in the presence of complement and dye.
- Lymphocyte destruction is evidenced by uptake of dye, indicating a positive crossmatch.
- A positive crossmatch is generally a contraindication to transplant as hyperacute rejection is likely.
- Panel Reactive Antibodies (PRA): PRA refers to the percentage of an antibody-screening panel with which the patient's serum reacts. High PRA means higher chances of rejection.

EXAM TIP

Graft survival is improved with matching for kidney, pancreas, and heart transplants, but donor-specific crossmatching does not have relevance for liver transplantation, as graft survival is not affected by positive crossmatch.

Immunosuppression

st er oids

- Most commonly prednisone (PO) or methylprednisolone (IV).
- Binds to intracellular receptor, is transported into nucleus—DNA-binding protein that works to limit, ultimately, the inf ammatory response by blocking nuclear factor-κB (NF-κB), interleukin-1 (IL-1), tumor necrosis factor-α (TNF-α), interferon, and phospholipase A_2, as well as histamine and prostacyclin.

EXAM TIP

Immunosuppression is associated with increased risk of infection and malignancies.

- Side effects: Impaired glucose tolerance, impaired wound healing, fluid retention, insomnia, depression, nervousness, and psychosis.
- Chronic effects: Cushing's syndrome, increased risk for peptic ulcer disease, osteoporosis.

Cyclosporine (CsA)

- Calcineurin inhibitor: CsA binds to cyclophilin and, together, they bind to calcineurin-calmodulin complex, blocking calcium-dependent phosphorylation and activation of NF-AT, thereby preventing transcription of several genes needed for T-cell activation, including IL-2. Put simply, it inhibits T-cell activity.
- P_{450} metabolism: Levels are increased by P_{450} inhibitors such as ketoconazole, erythromycin, calcium channel blockers, and decreased by P_{450} inducers like rifampin, phenobarbital, and phenytoin.
- Side effects: Nephrotoxicity (dose related, based on vasoconstriction of proximal renal arterioles), hemolytic-uremic syndrome (uncommon), hypertension, tremors, headache, paresthesia, depression, confusion, seizures (rare), hypertrichosis, gingival hyperplasia, and hepatotoxicity.

WARD TIP

Azathioprine is not generally used in liver transplant because it is likely to cause hepatotoxicity.

Mycophenolate Mofetil (MMf)

- Prodrug of mycophenolic acid (MPA): Noncompetitive reversible inhibitor of inosine monophosphate dehydrogenase—thereby halts purine metabolism, blocks proliferation of T and B cells, suppresses B-cell memory, and inhibits antibody formation.
- Side effects: Mild diarrhea or nausea.

WARD TIP

FK-506 and CsA have an additive immunosuppressive effect, but the toxicity may be too much.

Azathioprine

- Inhibits DNA synthesis by alkylating DNA precursors and depleting cell of adenosine.
- Side effects: Leukopenia, hepatotoxicity.

tacrolimus (fK-506)

WARD TIP

Some studies have found up to 40% incidence of posttransplant lymphoproliferative disorder with tacrolimus, and therefore, this drug is used with extreme caution in children.

- Drug of choice for liver transplant.
- Calcineurin inhibitor: Works in similar fashion to CsA, but binds initially to FK-binding protein.
- A hundred times more potent than CsA.
- Side effects as for CsA, though more neurotoxic and diabetogenic, and fewer cosmetic effects.

Antithymocyte globulin (Atg)

- Usually used for treatment of steroid-resistant rejection (SRR), but may also be used for induction therapy.
- Polyclonal antihuman γ-globulin extracted from horse sera of horses immunized with thymus lymphocytes.
- Side effects include fever and chills (20%), thrombocytopenia, leukocytopenia, rash (15%).

oKt 3

- Monoclonal antibody to CD3, a signal transducer on human T cells, thereby prevents transduction of antigen binding; also downregulates T-cell receptor (TCR).

- Used as rescue agent for SRR and also for induction.
- Side effects: Cytokine release syndrome (could lead to pulmonary edema/cardiac arrest).

Rejection

Hyper ACute

- **Cause:** Presensitization of recipient to donor antigen.
- **B cells** are responsible for antibody-mediated hyperacute rejection when the transplant contains an antigen that the recipient B cells have seen before.
- **Timing:** Immediately following graft reperfusion.
- **Mechanism:** Antibody binds to donor tissue, initiating complement-mediated lysis, which has a procoagulant effect. End result is thrombosis of graft.
- **Prevention:** ABO typing and negative crossmatch prevent hyperacute rejection in > 99% of patients.
- **Treatment:** None (graft removal).
- **Outcome:** Graft failure/loss.

ACute

- **Cause:** Normal T-cell activity (would ultimately affect every allograft were it not for immunosuppression).
- **Timing:** Between postoperative day 5 and postoperative month 6.
- **Mechanism:** T cells bind antigens in one of two ways—directly through TCR or after phagocytosis and presentation of donor tissue, resulting in T-cell infiltration of graft with organ destruction.
- **Diagnosis:** Generally by decreased graft function and by biopsy.
- **Histology:** Lymphocytic infiltrate and/or graft necrosis. Liver rejection also characterized by eosinophilic infiltrate.
- **Prevention:** Minimize mismatch of MHC; usual immunosuppression; monitor for organ dysfunction as signs of rejection that may otherwise be asymptomatic.
- **Treatment:** High-dose steroids; OKT3 or ATG when SRR after 2 days.
- **Outcome:** Ninety to 95% of transplants are salvaged with treatment.

CHroNiC

- **Cause:** Cumulative effect of recognition of MHC by recipient immune system.
- **Timing:** Insidious onset over months and years.
- **Mechanism:** Recipient's immune system recognizes donor MHC; other factors not yet understood.
- **Diagnosis:** Biopsy.
- **Histology:** Parenchymal replacement with fibrous tissue, some lymphocytic infiltrate, and endothelial destruction.
- **Prevention:** None known.
- **Treatment:** None.
- **Outcome:** Graft failure/loss.

riSK of MALigNANCy

- Overall incidence in kidney transplant recipients: 6%—lymphoma, skin cancer, genital neoplasms.

WARD TIP

Patients who are to receive OKT3 must be pretreated with steroids.

EXAM TIP

Presensitization of the recipient to a donor antigen can result from prior pregnancy, transfusion, or transplant.

EXAM TIP

Forty to 50% of kidney transplant recipients and 70–80% of pancreas transplant recipients will have at least one episode of acute rejection.

EXAM TIP

Typical scenario: A kidney transplant recipient is seen in the emergency department (ED) for nausea and abdominal pain, fever, and elevated creatinine. Think: Acute rejection. Diagnosis may be confirmed by ultrasound-guided biopsy. Pulse steroid treatment is indicated.

WARD TIP

Treatment of acute rejection of a kidney transplant (with pulse steroids or OKT3 for SRR) is effective in 90% of cases.

- Posttransplant lymphoproliferative disease (PTLD): Caused by Epstein–Barr virus (EBV), leads ultimately to monoclonal B-cell lymphoma/treatment to lower or stop immunosuppression and restore immunity.

EXAM TIP

Warm ischemia time should be minimized because it leads to rapid decline in adenosine triphosphate (ATP) and therefore decrease in biosynthetic reactions, a redistribution of electrolytes across cell membranes, and continuation of biodegradation reactions leading to acidosis and ultimately loss of organ viability.

Organ Preservation

Maximum times for each organ

- Heart: 4–6 hours.
- Lung: 4–6 hours.
- Liver: 8–12 hours.
- Pancreas: 12–18 hours.
- Kidney: 24–28 hours.

EXAM TIP

Morbidity and mortality associated with dialysis: A 49-year-old with end-stage renal disease (ESRD) on dialysis has an expected duration of life of 7 years, compared to 30 years for a healthy 49-year-old.

Kidney Transplantation

Background

Most common solid organ to be transplanted.

Causes of end-stage renal disease (esrd)

- Diabetes
- Hypertension
- Glomerular nephritis
- Congenital anomalies
- Urologic abnormalities
- Dysplasia
- Focal segmental glomerular sclerosis

EXAM TIP

Decreased quality of life in ESRD is associated with time commitment for dialysis and increased number of hospital days.

types of donors

- **Cadaveric.**
- **Living:**
  - May be related or unrelated.
  - Decreased warm ischemia time.
  - Associated with less delayed graft function and better outcome.
  - Donor mortality: 0.02–0.03%.
  - Living donor evaluation: Rule out potential donors with:
    - Diabetes, hypertension, malignancy, coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), renal disease, human immunodeficiency virus (HIV), hepatitis B/C, age < 18.
    - Genitourinary (GU) anomalies assessed by proteinuria > 300 mg/24 hr or creatinine clearance rate (CCr) < 80 mL/min, urologic abnormalities of donor kidney.

EXAM TIP

Cardiovascular disease is responsible for 50% of dialysis patients' deaths, and infection accounts for 15–30%.

Appropriate recipient

- General health assessment:
  - Identify comorbidities such as heart disease, COPD, and diabetes.
  - Assess ability to handle immunosuppression and compliance.

TABLE 25-1. Contraindications to Kidney Transplantation

Absolute Contraindications	Relative Contraindications
Cancer (other than squamous cell or basal cell carcinoma of skin)	Sickle cell disease
Infection (HIV, tuberculosis)	Likely to be noncompliant
Cirrhosis (chronic active)	Ischemic heart disease severe, without possibility of coronary artery bypass graft or angioplasty
Ongoing drug use	

- Laboratory evaluation consisting of chemistries, complete blood count (CBC), urinalysis, serologies for hepatitis B and C, cytomegalovirus (CMV), and HIV.
- Conduct ECG and chest x-ray (CXR).
- Further cardiac workup based on need.
- Psychosocial evaluation regarding compliance.
- May need bilateral nephrectomy for recurrent urinary tract infections (UTIs) or polycystic kidney disease (PCKD).
- See Table 25-1 for contraindications to kidney transplantation.
- The donor kidney is usually placed in the iliac fossa with vascular anastomosis to the iliacs and the native kidney of the recipient need not be removed excepting for PCKD and recurrent UTIs.

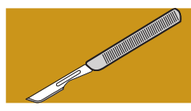
COMPLICATIONS SPECIFIC TO KIDNEY TRANSPLANTATION

Early Complications

- Delayed graft function.
- Evidenced by oliguria or anuria (assess volume status, ensure that Foley is working; once checked, but still anuric/oliguric, Doppler ultrasound [US] indicated to assess blood flow). If blood flow is adequate, look for urine leak or obstruction at ureterovesicular junction (UVJ) with US or renal scan. Once all workup negative, diagnosis is delayed graft function.
- Management may include dialysis in postoperative period.
- **Graft thrombosis:**
  - Requires immediate reoperation to save transplant.
  - Diagnosis indicated by abrupt cessation of urine output.
  - May assess with Doppler US.
- **Urine leak:**
  - Usually at UVJ.
  - **Diagnosis:** Decreased urine output, lower abdominal pain, scrotal or labial edema, rising creatinine.
  - **Tests:** US with fluid aspiration and analysis, renal scan with extravasation of radioisotope.
  - **Treatment:** Reexploration and repair.

Late Complications

- **Ureteral stricture:**
  - Rising creatinine and hydronephrosis on US.
  - Distal stricture result of rejection or ischemia; antegrade pyelogram best diagnostic tool.
  - **Treatment:** Balloon dilatation; longer ones require surgical repair.



OR TIP

The left kidney is preferred by surgeons because of its longer renal vein, but preoperative imaging studies in the potential donor can identify variants of normal anatomy (like multiple arteries) that may make the right kidney a better choice.

- Renal artery stenosis:
  - Ten percent of renal transplants within first 6 months.
  - **Presentation:** Hypertension, fluid retention.
  - **Diagnosis:** Angiogram, US, magnetic resonance angiography (MRA).
  - If distal to anastomosis, may be secondary to rejection, atherosclerosis, clamp or other iatrogenic injury. Occurs more frequently with end-to-end anastomoses.
  - **Treatment:** > 80% correctible with angioplasty; others require surgical repair.
  - **Donor complications:** UTI, wound infection, pneumothorax.

Pancreas Transplantation

Indications

- Type 1 (insulin-dependent) diabetes mellitus.
- Intensive insulin therapy may slow the rate of secondary complications of diabetes, but at increased risk of hypoglycemic episodes.

WARD TIP

Alternatives to whole-organ pancreas transplant are use of insulin pump and islet of Langerhans transplant.

Exclusions for Recipients

- Significant CAD.
- Severe peripheral vascular disease (PVD) resulting in amputations.
- Severe visual impairment.
- Untreated malignancy.
- Active infection.
- HIV.
- **Exclusion criteria for donors:**
  - Pancreatic disease and type 1 DM.

OR TIP

The pancreas transplant is placed in the abdominal cavity, preferably on the right side rather than in the retroperitoneal space because of a lower incidence of peripancreatic fluid collections and lymphocele.

Timing of Operation

- **Pancreas transplant (5%):** For the nonuremic diabetic patient with minimal or no evident nephropathy.
- **Pancreas transplant after kidney transplant (PAK) (15%):** Previously underwent kidney transplant, but now have poor glucose control or progression of diabetes complications.
- **Simultaneous pancreas and kidney transplants (SPK) (80%):** In diabetic and uremic patients.

WARD TIP

pancreas transplant: Expect euglycemia within 12 hours of operation; no postop insulin should be required.

Drainage Options

- **Bladder drainage:**
  - Advantage: Urinary amylase may be used as a sign of rejection; rejection of exocrine pancreas precedes the endocrine pancreas.
  - Up to 25% may require conversion to enteric drainage.
- Avoids bacterial contamination.
- **Enteric drainage:**
  - Avoidance of postop GU complications that affect 30% of bladder-drained patients.
  - Avoidance of chronic dehydration.
  - No need for bicarbonate replacement.
  - Equal efficacy, graft survival, morbidity.

WARD TIP

Consider acute rejection of a pancreas transplant when urinary amylase is consistently decreased by 25%.

- In SPK, rejection usually involves pancreas and kidney. May diagnose by creatinine and renal biopsy.
- Enteric drainage more common in US.

Complications (specific to pancreatic transplant)

- Thrombosis, venous more common than arterial
- Bleeding
- Intra-abdominal abscess
- Pancreas specific complications like pancreatitis/pseudocysts/fistulas
- If bladder drainage has cystitis/hematuria/metabolic acidosis/diarrhea/dehydration

Liver Transplantation

Indications

- Irreversible liver failure.
- **Chronic (more common):**
  - Cirrhosis (posthepatitic/alcoholic).
  - Primary and secondary biliary cirrhosis.
  - Primary sclerosing cholangitis.
  - Metabolic defects (α_1-antitrypsin deficiency, amyloidosis, hemochromatosis, sarcoidosis, tyrosinemia, ornithine transcarbinase deficiency, Crigler–Najjar).
  - Malignancy (hepatocellular carcinoma [HCC] or cholangiocarcinoma).
  - Others: Biliary atresia, cystic fibrosis.
- **Acute or fulminant:**
  - Viral or alcoholic hepatitis.
  - Wilson's disease.
  - Hepatotoxic drugs (e.g., acetaminophen overdose).
  - Contraindications.
  - Multisystem organ failure/life-threatening systemic disease.
  - Severe cardiopulmonary disease.
  - Sepsis secondary to nonhepatic source.
  - Metastatic malignancy.
  - Noncompliance with medical therapy/active drug/alcohol abuse.
  - Severely impaired neurologic status.

types of liver transplantation

- Cadaveric: Can also be split between two recipients
- Living donor transplantation

evaluation

- The Model for End-Stage Liver Disease (MELD) score is used for adult liver transplant assessment (uses patient's values for bilirubin, international normalized ratio [INR] for prothrombin time, and creatinine). Also takes into consideration if patients are being dialysed.
- It is a numerical score from 6 to 40 and gives a score based on how urgently patient needs liver transplant in next 3 months.
- Preoperative control of:
  - Variceal bleeding: Transjugular intrahepatic portosystemic shunt (TIPS) when needed.
  - Ascites: Diuretics and/or paracentesis.

WARD TIP

Bladder irrigation may help clear up hematuria and prevent clot retention.

EXAM TIP

UTI is the most common infectious complication after pancreas transplants, especially following bladder-drained SPK transplants.

WARD TIP

Liver transplant outcome is generally better for chronic liver failure patients.

WARD TIP

Cancer and alcohol abuse remain controversial indications for liver transplant:

- Alcoholic liver disease accounts for 75% of liver failure in the United States. Patients have excellent outcome, with low rate of recidivism. Pretransplant abstinence, evidence of support system, and lack of other alcohol-related comorbidities are required.
- Patients transplanted for liver cancer have worse outcome than patients transplanted for other reasons, but survival is improved compared to cancer patients who are not transplanted (40% 5-year survival).

Complications

- Graft failure: Usually secondary to primary nonfunction, recurrence of disease, biliary or vascular complications (not generally due to rejection).
- Systemic complications like renal failure, cardiopulmonary complications.
- Rejection occurs in first 3 months posttransplant with 50% incidence, but is well treated with steroids or antilymphocyte therapy (indicated by elevated liver function tests [LFTs], particularly γ-glutamyl transpeptidase [GGTP]).

Small Bowel Transplantation

Indications

Life-threatening complications due to intestinal failure and complications related to parenteral nutrition.

- Adults: Short bowel syndrome, due to Crohn's disease, mesenteric thrombosis, trauma.
- Children: Short bowel syndrome, due to necrotizing enterocolitis, intestinal pseudo-obstruction, gastroschisis, volvulus, intestinal atresia.

Operation

- Isolated intestinal failure: Isolated intestinal transplant.
- With liver failure: Liver-intestine combined transplant.
- Sometimes, multivisceral transplant: Liver, pancreas, duodenum, small intestine.
- Stoma usually placed for monitoring and biopsies.

Complications

- Graft-versus-host disease (GVHD): Prevent with immunosuppression and/or pretreatment of donor.
- Rejection: More difficult to treat than in other organs; newer agents may prove to be more useful than older ones (tacrolimus based).
  - CMV infection
- Diagnosed by fever, abdominal pain, elevated white count, ileus, gastrointestinal (GI) bleed, positive blood cultures; also by biopsy showing cryptitis, villi shortening, mononuclear infiltrate.

Cardiac Transplantation

Indications

- Severe cardiac disability on maximal medical therapy (multiple hospitalizations for congestive heart failure [CHF], New York Hospital Association class III [NYHA III] or IV, or peak oxygen consumption < 12–14 mL/kg/min).
- Symptomatic ischemia or recurrent ventricular arrhythmias refractory to usual therapy, with left ventricular ejection fraction (LVEF) < 30%, or with unstable angina and not a candidate for CABG or percutaneous transluminal coronary angioplasty (PTCA).
- Severely symptomatic hypertrophic or restrictive cardiomyopathy
- All surgical alternatives already excluded.

Contraindications

- Irreversible, severe, pulmonary hypertension, renal, or hepatic dysfunction.
- Unstaged or incompletely staged cancer.
- Active infection.
- Psychiatric illness.
- Severe systemic disease.

Evaluation

Matching/compatibility based on:

1. ABO compatibility.
2. Body size.
3. HLA crossmatching for presensitized heart transplant recipients.

Complications

- The most common cause of perioperative death is infection (50%). Other common causes are pulmonary hypertension and nonspecific graft failure.
- **Infection:**
  - Peak incidence: Early postoperative period.
  - Most patients will have an infection at some point in the first five post-operative years.
- **Rejection:**
  - At least 75% incidence in first three posttransplant months.
  - Diagnosis confirmed by endomyocardial biopsy (via right internal jugular).
- **Histology:** Lymphocyte infiltration and/or myocytic necrosis.
- **Treatment:** Pulse steroids if early on (later in course, mild rejection treated by 3-day increased dose of oral prednisone); refractory rejection treated with ATG or OKT3.
- **Chronic rejection:** Concentric intimal proliferation with smooth muscle hyperplasia yielding atherosclerosis more diffuse than in nontransplanted hearts.
- Prevalence of CAD: 25% at 1 year and upto 50% at 5 years. Because of denervation, patients do not develop angina.
- Yearly angiogram recommended. Alternative for surveillance is intravascular US.
- **Cancer:** Increased risk, with immunosuppression, of skin, vulvar, or anal cancer; B-cell lymphoproliferative disorder (BLPD) and cervical intraepithelial neoplasia (CIN).

Lung Transplantation

Types of Operations and Indications

- Single lung for fibrotic lung disease: Pulmonary fibrosis, emphysema, bronchopulmonary dysplasia, primary pulmonary hypertension (without cardiac dysfunction), posttransplant obliterative bronchiolitis.
- Bilateral for cystic fibrosis, bronchiectasis, COPD.
- Heart-lung for pulmonary vascular disease, end-stage lung disease with cardiac dysfunction.
- Lobar lung, to increase donor pool from living related and cadaveric donors.

WARD TIP

Typical scenario: A 53-year-old woman who is status post liver transplant calls your office asking what she can take for some musculoskeletal pain. Think: Patient is on tacrolimus, which ultimately causes renal insufficiency in most patients. Do not give anything that could potentiate its nephrotoxicity. First-line drug for pain would be acetaminophen, standard doses of which a transplanted liver should be able to tolerate.

OR TIP

Cardiac transplant: Cold ischemic time up to 6 hours may be tolerated, though 3–4 is ideal (2 hours maximum for patients with pulmonary hypertension).

WARD TIP

Cardiac graft vasculopathy is a major cause of late graft failure. It is an aggressive form of atherosclerosis.

WARD TIP

Transplanted heart has increased sensitivity to catecholamines, with increased density of adrenergic receptors with loss of norepinephrine uptake; cardiac output and index remain at low normal, with adequate but abnormal exercise response (increase in heart rate is usually delayed).

WARD TIP

Patients have increased risk of cholelithiasis and strong consideration should be given to pre and posttransplant surveillance.

Contraindications

- Significant systemic disease, including hepatic or renal disease.
- Active infection.
- Malignancy.
- Noncompliance.
- Current smoking.
- Chronic hepatitis B/C and HIV.

Evaluation

- ABO compatible.
- Infection-free donor and recipient.
- Donor: Good pulmonary gas exchange, no smoking history, similar lung volume, where possible.

EXAM TIP

CMV occurs at 75–100% incidence in cardiac transplant patients and has been identified as trigger for graft-related atherosclerosis (treated with ganciclovir and hyperimmune globulin).

Complications

- **Acute rejection:**
  - Sixty to 70% incidence in first postoperative month.
  - Diagnosed by clinical parameters: Fever, dyspnea, decreased Pao_2, decreased forced expiratory volume in 1 second (FEV_1), CXR with interstitial infiltrate.
  - Confirm with biopsy via bronchoscopy: Lymphocytic infiltrate to varying degrees.
- **Infection:** Early, predominance of gram-negative bacillus (GNB) infection.
  - **Viral:** CMV, treated with ganciclovir, also EBV.
  - **Fungal:** Candida, Aspergillus.
  - **Protozoan:** Pneumocystis jiroveci pneumonia.
- **Outcomes:**
  - Kidney transplants have best outcomes, 5-year graft survival for living donor is 81% (90% patient survival) in the United States.
  - Small bowel and lung have the lowest rate of organ survival.

EXAM TIP

Chronic rejection manifests as bronchiolitis obliterans, seen in upto 50% after 5 years. Dense eosinophilic scarring of bronchioles. No proven beneficial treatment.

HIGH-YIELD FACTS IN

The GeniTourinary SySTem

Anatomy of the Genitourinary (GU) System 414
Penis 414
Scrotum 414
testes 414
ePididymis 415
Vas deferens 415
Prostate 415
Kidneys 415
ureters 415
Bladder/urethra 415

Hematuria 416
definition 416
causes 416
other causes 416

Priapism 417

Acute Scrotum 417
Signs and SymPtoms 417

Testicular Torsion 418
PathoPhysiology 418
Signs and SymPtoms 418
diagnosis 418
treatment 419

Testicular Tumor 419
definition and ePidemiology 419
riSKfactors 419
Signs and SymPtoms 419
diagnosis 419
classification and Pathology 419
Staging eValuation 420
treatment 420

Urolithiasis 420
definition and ePidemiology 420
etiology 421
riSKfactors 421
Signs and SymPtoms 421
diagnostic tests 421
management 422

Benign Prostatic Hyperplasia 422
definition 422
PathoPhysiology 423
Signs and SymPtoms 423
diagnosis 423
management 423

Prostatic Carcinoma 423
definition 423
riSKfactors 423
classification 424
Signs and SymPtoms 424
diagnosis 424
Staging 424
treatment 425

Renal Cell Carcinoma (RCC) 425
etiology 425
Signs and SymPtoms 425
diagnosis 425
Staging 426
treatment 426
Prognosis 426

Urothelial Cancer 426
definition and ePidemiology 426

Transitional Cell Carcinoma of the Collecting System and Ureters 426

Bladder Cancer 426
Signs and SymPtoms 427
diagnosis and Staging 427
treatment 427
Prognosis 428

Anatomy of the Genitourinary (GU) System

Penis

- Made up of three cylindrical bodies (see Figure 26-1):
  - Two corpora cavernosa covered by tunica albuginea, comprising the erectile tissue of the penile shaft.
  - One corpus spongiosum, which surrounds the urethra and expands to form the glans.
  - Buck's fascia surrounds the corpora cavernosa and corpus spongiosum structures.
- Blood supply: Internal pudendal artery. Dorsal veins drain into the pudendal plexus, which in turn drains into pudendal vein.
- Lymphatic drainage: Deep and superficial inguinal nodes.

EXAM TIP

Cryptorchidism increases the risk for cancer. Approximately 10% of testicular tumors (usually seminoma) arise from undescended testes.

EXAM TIP

Bell-clapper deformity: Insufficient attachment of the testicles to the tunica vaginalis, allowing for a pathologically large degree of rotational freedom. This increases risk of torsion.

scrotum

- Made up of smooth muscle, elastic tissue layers of Darto's fascia, which is a continuation of Scarpa's fascia from the abdominal wall.
- Blood supply: Femoral and internal pudendal artery.
- Lymphatic drainage: Inguinal and femoral nodes.

testes

- Lie in the scrotum, usually vertically.
- Encased in tunica albuginea except posteriolaterally, where it is attached to the epididymis. This entire unit is then wrapped in the tunica vaginalis, which attaches to the scrotum posterior-inferiorly via the gubernaculum.
- Vestigial structures: The appendix testis is located in the superior aspect of the testicle, and is of Müllerian duct origin. The appendix epididymis is located on the epididymis, and is of Wolffian duct origin.

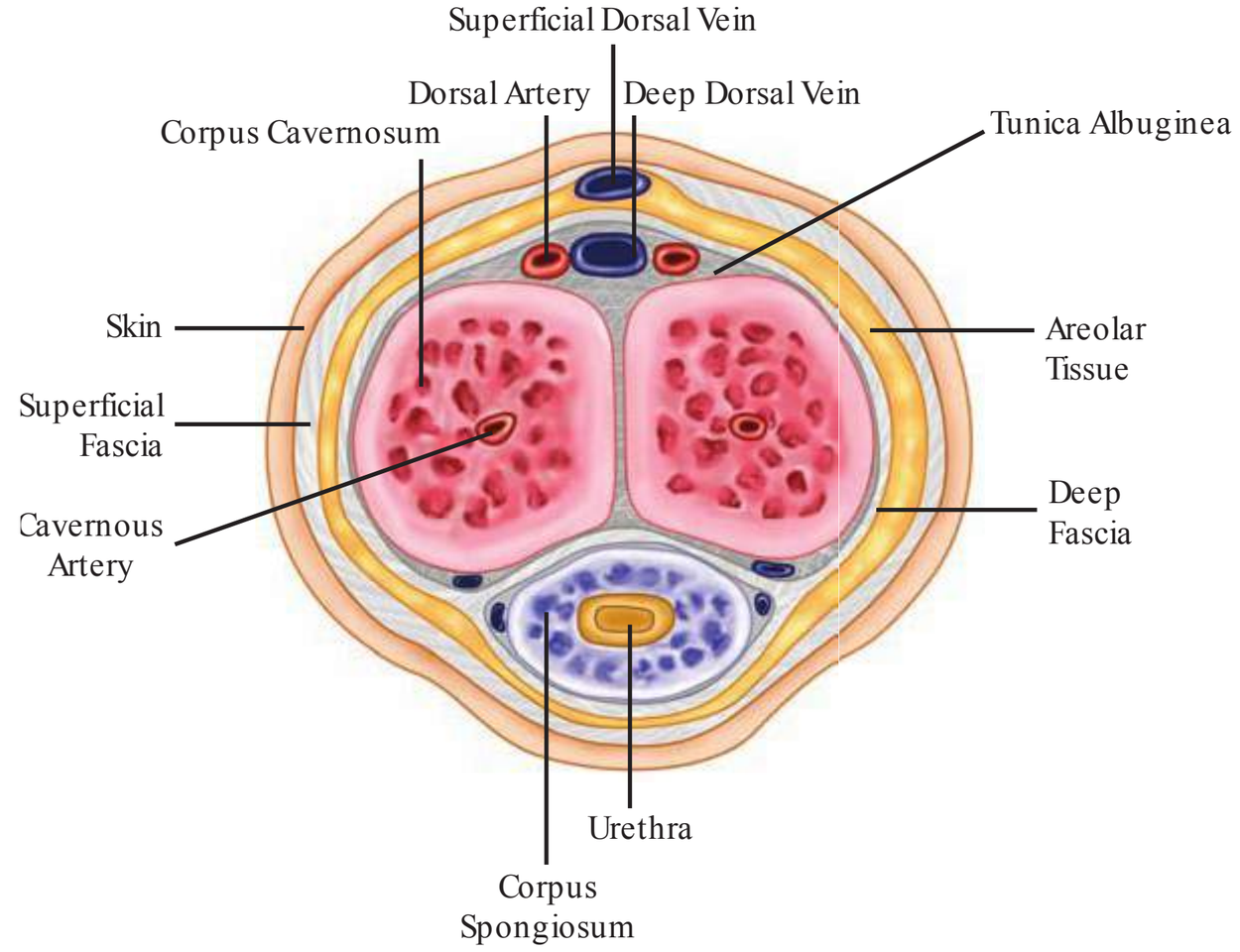


FiGure 26-1. Cross-section of the penis. (Reproduced, with permission, from Le T, Krause K. First Aid for the Basic Sciences: Organ Systems. New York: McGraw-Hill; 2009: 753.)

- Blood supply to testis: Gonadal artery, deferential artery, and cremasteric vessels. All three travel in the spermatic cord!
- Venous return: Pampiniform plexus, which in turn drains into the gonadal vein and, to a lesser extent, the external pudendal, vasal, and cremasteric veins.
- Lymphatic drainage: External, common iliac, and periaortic nodes.

ePididymis

- Tubular structure, originating from the Wolffian duct.
- Four to 5 m in length compressed to an area of about 5 cm.
- Site of sperm maturation and motility.

Vas deferens

- Originates from the Wolffian duct.
- A muscular tube that originates at the epididymis, exiting the scrotum laterally. It then courses into the inguinal canal via the external ring and exits the canal at the internal ring. Here, the vas deferens diverges from the spermatic cord, crossing medially behind the bladder and over the ureters to form the ampulla of vas deferens. It then joins the seminal vesicles to form the paired ejaculatory ducts in the prostatic urethra.
- Spermatic cord contains vas deferens, testicular vessels, cremasteric muscle fibers, and spermatic fasciae.

Prostate

- Originates from the urogenital sinus and matures via dihydrotestosterone stimulation.
- Secretory gland that contributes to seminal fuid, along with seminal vesicles, Cowper's glands, and epididymis.
- Posterior surface easily palpable on digital rectal exam (DRE).

Kidneys

- Kidneys, like all urologic organs, are retroperitoneal structures.
- Lie obliquely on psoas muscle and quadratus lumborus.
- Renal hilum consists of (from anterior to posterior) vein, artery, and ureter/renal pelvis. Occasionally, there is an accessory artery elsewhere. On the left side, the gonadal vein inserts perpendicularly into the renal vein. However, on the right side, the gonadal vein inserts obliquely directly into the inferior vena cava (IVC) (Figure 26-2).
- Surrounded by perirenal fat, both the kidneys and adrenal glands lie within Gerota's fascia.
- The adrenal gland is located superior-medially to the kidney. However, in cases of renal ectopia and agenesis, the adrenal gland remains orthotopic.

ureters

Ureters course along the psoas muscle, crossing the iliacs anteriorly at the bifurcation of the external/internal iliac arteries. The ureters then course medially toward the bladder, inserting posteriorly.

Bladder/urethra

- Bladder consists of detrusor muscle, covered by a urothelial layer. Histologically, this is transitional epithelium.

WARD TIP

Clinical Pearl: Varicoceles are far more common on the left, due to the difference in drainage pattern of each side's gonadal vein (see Kidney section below).

EXAM TIP

Any testicular mass that is solid (i.e., does not transilluminate), is not biopsied, but testicle is entirely excised (radical orchiectomy). This is performed through an inguinal incision, not a scrotal incision.

WARD TIP

Torsion of the testicular or epididymal appendix can cause extreme pain mimicking torsion of the testes. However, this is a benign entity and is treated with NSAIDs.

WARD TIP

Obstruction at the renal pelvis is a common cause of hydronephrosis. This may be caused by a crossing blood vessel to the kidney or a non-gravity-dependent ureteral insertion into the renal pelvis.

EXAM TIP

A pathologically short intramural ureter predisposes a patient to vesicoureteral reflux.

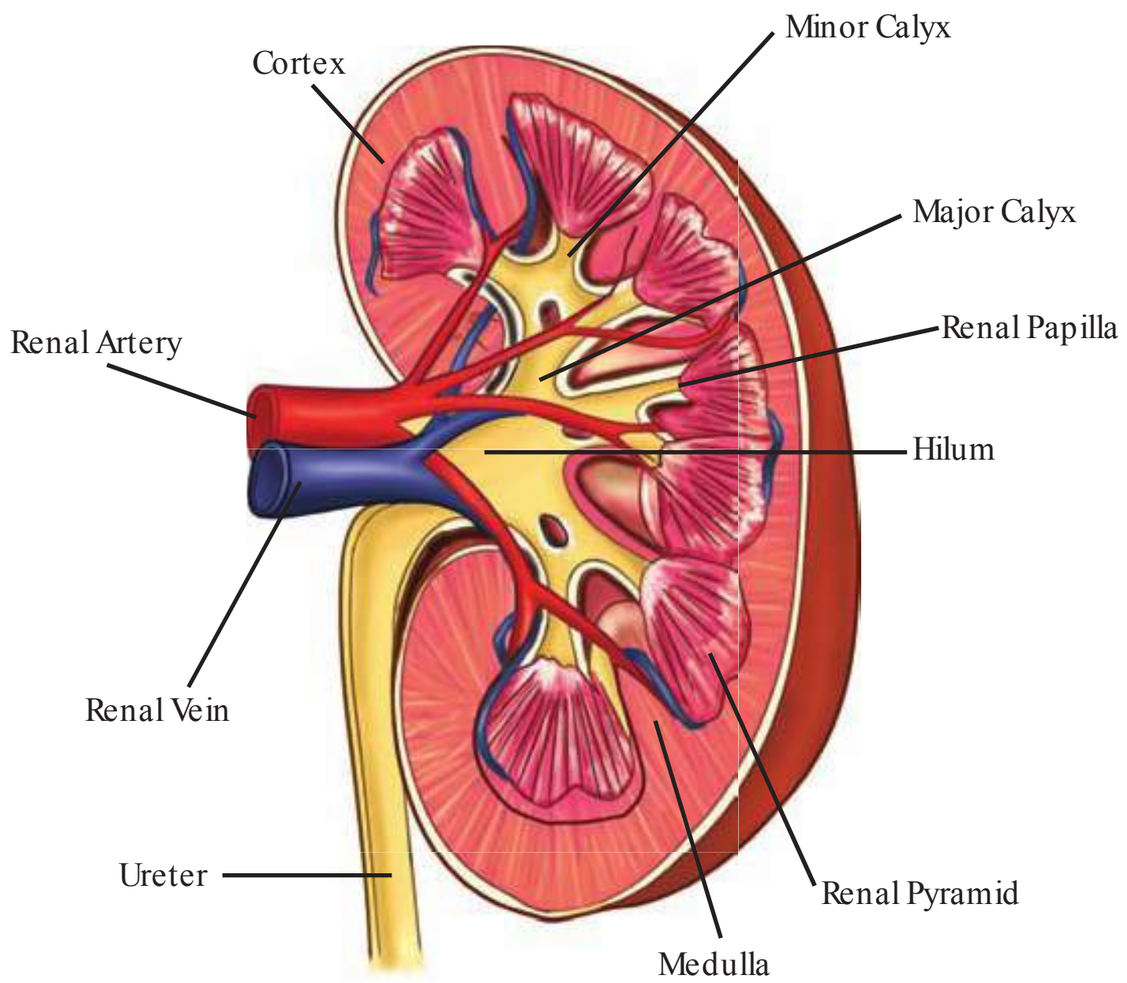


FiGure 26-2. Gross anatomy of the kidney. (Reproduced, with permission, from Le T, Krause K. First Aid for the Basic Sciences: Organ Systems. New York: McGraw-Hill; 2009: 656.)

- Both the detrusor and the bladder neck (internal sphincter) are smooth muscle with autonomic innervation. The external (striated) urinary sphincter is under voluntary control.
- In males, the prostatic urethra is located between the internal and external sphincters (this is a common site of adenomatous growth and urinary obstruction).

Hematuria

definition

- Presence of blood in the urine.
- Can be gross or microscopic.
- ≥ 3 red blood cells (RBCs) per high power field of spun urine.

EXAM TIP

In working up microhematuria, assess for the presence of red cell casts, which are present in glomerular bleeding.

causes

- **Pediatric:** Acute urinary tract infection (UTI), glomerular origin, congenital urinary tract abnormality, stones, trauma.
- **Adult/elderly:** Acute UTI, bladder cancer, urolithiasis, benign prostatic hyperplasia.

other causes

- **Upper tract:** Sickle cell disease, collagen vascular disease, renal disease (glomerulonephritis, vascular abnormalities, pyelonephritis, polycystic kidney, granulomatous disease, interstitial nephritis, neoplasm/renal cell carcinoma [RCC]).
- **Lower tract:** Urethritis, stones, cystitis, prostatitis, epididymitis. Mild hematuria is a normal finding in a patient with recent urologic instrumentation (including catheterization).

EXAM TIP

False positive on dipstick occurs when myoglobin is present.

- Coagulopathy and anticoagulation may unmask (but not cause) hematuria and a proper workup should be considered regardless of the etiology or level of coagulopathy.

Priapism

- **Definition:** An erection lasting more than 4 hours, generally not maintained by sexual stimulation.
- Two types: Diagnosed by history and physical and intracorporeal arterial blood gas (ABG)—see below.
  - **High flow:** Not ischemic (pH ~7.4), not painful. May result from arteriovenous (AV) fistula secondary to trauma (usually prior injection/irrigation as treatment for low flow).
- May be erect for days to weeks. Erection not fully turgid and glans generally not engorged. Treated with angioembolization of AV fistula site. Less common than low flow.
- **Low flow:** More common. Painful and entirely erect. Ischemic (pH < ~7.2), so risk of long-term erectile dysfunction, corporal fibrosis, or organ loss in severe cases. Common causes: Intracorporeal Caverject (alprostadil) injections, cocaine, trazadone, PDE5 inhibitors (e.g., sildenafil), sickle cell.
- Initial treatment involves local ice application, IM verapamil and Sudafed. If these fail (and they will!), patients require intracorporeal phenylephrine injections with or without saline irrigation of the corporal. (Remember to monitor hemodynamics closely when giving intra-corporeal phenylephrine injections!) All of the above can be used when treating sickle cell priapism, but systemic treatments should be first attempted (i.e., exchange blood transfusion, oxygen administration, aggressive hydration hydroxyurea).

EXAM TIP

What can cause gross hematuria with a dipstick negative for blood? Think: Anthocyanin dye in beets and berries, pyridium, rifampin, porphyria, some food colorings.

Acute Scrotum

- **Differential diagnosis:** Testicular torsion, epididymo-orchitis, testicular tumor, peritesticular tumor (rhabdomyosarcoma, leiomyosarcoma, liposarcoma), torsion of testicular appendage, orchitis, trauma to scrotum, hernia, hydrocele, varicocele.
- Testicular torsion is a surgical emergency and must always be considered in the case of acute scrotal pain or mass.
- The diagnosis of an acute scrotal mass will depend largely on the history and physical examination.
- A hydrocele is a collection of fluid between the testicle and the tunica vaginalis. It is typically painless; however, it may occur in response to an inflammatory testicular process.

WARD TIP

Whereas adult hydroceles gradually enlarge, pediatric hydroceles grow throughout the day and typically disappear overnight as the fluid flows back through the processus vaginalis into the peritoneal cavity.

signs and symptoms

Important findings to look for in acute scrotal mass:

- **Lateralization of swelling.**
- Erythema of scrotal skin.
- Position of the testicle.
- Localization of testicular mass and pain.
- Presence of hydrocele by transillumination.
- Presence/absence of cremasteric reflex.

WARD TIP

Transillumination can easily distinguish a hydrocele from other testicular masses.

WARD TIP

The cremasteric reflex is elicited by stroking the medial aspect of the patient's thigh, resulting in a contraction of the cremasteric muscle elevating the scrotum and ipsilateral testis.

- Prehn's sign—relief of pain by elevation of the testicle; indicative of epididymitis.
- Urethral discharge, as well as inguinal lymphadenopathy.
- Fever.
- Recent history of trauma.
- History of similar pain.
- Acute vs. subacute onset.
- Previous history of urethral discharge, sexually transmitted infections, or unprotected sex.

Testicular Torsion

WARD TIP

Always suspect torsion in a patient with inguinal/abdominal pain and an empty scrotum.

- Peak incidences during the first year of life and at puberty (when growth rates are the fastest).
- Undescended testis confers a higher risk of ipsilateral torsion.
- Salvage rate is 80–100% if surgical intervention is < 6 hours from the onset of pain.
- Frequently preceded by strenuous activity or athletic event.
- Acute onset.

PathoPhysiology

EXAM TIP

Typical scenario: An adolescent presents with acute testicular pain and swelling immediately after a sporting event. He is writhing in pain. On further questioning, he has had similar episodes of this in the past. Think: Intermittent testicular torsion/detorsion.

- The testicle twists on its spermatic cord, causing obstruction of venous return leading to swelling. If obstruction persists, venous thrombosis occurs, followed by arterial occlusion. Infarction ensues quickly in the absence of blood supply.
- "Bell-clapper deformity" (see above).

signs and symPtoms

- Acute onset of severe pain in testicle, lower abdomen, or the inguinal canal.
- Scrotum is elevated, swollen, and tender.
- May be accompanied by nausea or vomiting.
- Classic sign: High-riding testis with a horizontal lie. However, this is often difficult to distinguish because of the degree of swelling in the scrotum.
- Presence of a reactive hydrocele.
- Loss of cremasteric ref ex.

diagnosis

EXAM TIP

The higher the undescended testicle, the higher the lifelong risk of cancer (irrespective of whether or when the testicle is brought into the scrotum surgically).

- Since epididymitis is most commonly confused with testicular torsion, several elements may aid in distinguishing the two entities. In torsion:
  - Urinalysis (UA) and complete blood count (CBC) will be unremarkable.
  - Onset is acute and often recurrent.
  - Most will be afebrile.
- Since the ability to salvage the affected testis is dependent on how quickly the testis becomes detorsed, time is testis! If the diagnosis is thought to be torsion, surgical exploration is indicated.
- Color Doppler ultrasound (US) or nuclear scintigraphy have become the tests of choice, showing the presence or absence of blood f ow to the suspected testis.

treatment

- Surgical repair is the definitive treatment.
- If the testis is viable, bilateral orchiopexy is performed. If the tissue is necrotic, the testicle is removed to prevent gangrene. The contralateral testis undergoes orchiopexy as a prophylactic measure.

Testicular Tumor

definition and epidemiology

- The most common malignancy to affect young men.
- There is a peak frequency in early childhood, and a larger peak incidence between 20 and 35 years of age. Uncommon after age 40.
- Occurs in whites more than African-Americans.

risk factors

- Men with cryptorchid (undescended) testes (intra-abdominal testes with the highest risk). It is important to note that both testicles are at risk.
- Surgical placement of the testis into the scrotum does not decrease malignant risk, but facilitates surveillance.
- Testicular cancer in the contralateral testis.

signs and symptoms

- Painless enlargement of the testicle.
- Firmness of the testicle.
- Back or abdominal pain secondary to retroperitoneal (intra-aortocaval) lymphadenopathy.
- Weight loss.
- Dyspnea secondary to pulmonary metastasis.
- Gynecomastia secondary to hormonal secretions of the tumor.

diagnosis

- Physical examination.
- Testicular US.
- CT scan/magnetic resonance imaging (MRI) of abdomen and pelvis to assess for metastasis and lymphadenopathy.
- Tumor markers—α-fetoprotein (AFP), human chorionic gonadotropin (hCG), and lactic dehydrogenase (LDH).
- Roughly 10% of seminomas secrete hCG; 90% of seminomas do not have elevated tumor markers.

classification and Pathology

- Classification is based on the cell type from which the tumor is derived—germinal or stromal.
- Germinal cell tumors comprise 95% of all testicular tumors:
  - Seminomas (pure single-cell tumors) comprise 35% of testicular tumors.
  - Nonseminomas (embryonal cell carcinoma 20%, teratoma 5%, choriocarcinoma < 1%).
  - Combination tumors 40%.

EXAM TIP

The prognosis of seminomas is excellent due to its exquisite sensitivity to radiation!

- Tumors of gonadal stroma (1–2%):
  - Leydig cell.
  - Sertoli cell.
  - Primitive gonadal structures.
  - Gonadoblastomas (germinal cell + stromal cell).

staging eValuation

- To determine whether the cancer is localized to the testis, regional lymphatics or widely metastasized.
- Abdominal and pelvic CT scan to determine the presence of adenopathy or visceral involvement, chest x-ray (CXR) ± chest CT.
- There are many staging systems; however, most include three stages:
  - **Stage I:** No clinical or radiographic evidence of tumor presence beyond the confines of the testis. Markers normalize after orchiectomy.
  - **Stage II:** Retroperitoneal adenopathy on CT scan, subdiaphragmatic disease, and failure of markers to normalize after orchiectomy.
  - **Stage III:** Distant metastases or visceral involvement.

treatment

- **Surgical approach:** High radical inguinal orchiectomy.
- Trans-scrotal biopsy of the testis or a trans-scrotal orchiectomy should not be performed if the diagnosis of testicular cancer is suspected because the lymphatic drainage of the testes is different from that of the scrotum; a scrotal incision in the presence of testicular cancer may cause local recurrence, metastasis, and unpredictable lymphatic spread.
- **Early seminoma:** Orchiectomy + retroperitoneal x-ray therapy (XRT).
- **Advanced seminoma:** Orchiectomy and combination chemotherapy followed by restaging.
- **Stage I nonseminoma:** Orchiectomy + retroperitoneal lymph node dissection (RPLND) or surveillance.
- **Stage II nonseminoma:** The optimal management of this group of patients is controversial. RPLND can be curative but have a high relapse rate. If relapse occurs, chemotherapy can be given as adjunctive therapy. Alternatively, chemotherapy can be given prior to RPLND.
- Advanced stage nonseminoma: Orchiectomy + chemotherapy ± tumor reductive surgery.
- The most commonly used chemotherapeutic regimen: BEP (etoposide, bleomycin, cisplatin).

Urolithiasis

definition and ePidemiology

- One of the most common diseases of the urinary tract.
- Male-to-female ratio of 3:1.
- Familial tendency in stone formation.
- Average risk is 1%/year.
- Tendency for recurrence: Thirty-six percent of patients with a first stone will have another stone within one year, and 50% will recur within years.

etiology

- Most common are calcium oxalate or a mixture of calcium (75%).
- Magnesium-ammonium-phosphate stones (struvite) 15%—seen in UTI with urea-splitting bacteria (Proteus). May cause staghorn calculi.
- Uric acid stones (15%), which are radiolucent (will not show up on x-ray, but are visible on noncontrast CT).
- Less than 1% are cystine stones (secondary to an inborn error of metabolism).
- Indinavir stones are not visible on CT scan or plain films.

risKfactors

- Poor fuid intake/residence in a hot climate.
- Disease processes that increase free calcium, including bony destruction, multiple myeloma, hyperparathyroidism, osteolytic lesions, and sarcoidosis.
- Prolonged immobilization.
- History of calculus in the past and in family members.
- Drug ingestion (analgesics, alkalis, uricosuric agents, protease inhibitors).
- Prior history of gout/Lesch–Nyhan.
- Underlying gastrointestinal (GI) disease/malabsorption (Crohn's, ulcerative colitis, peptic ulcer disease [PUD], gastric bypass surgery).
- In a normal individual, dietary calcium intake is **not** a risk factor.

signs and sympt oms

- Severe, abrupt onset of colicky pain, which begins in the fank and may radiate toward the groin. In males, the pain may radiate toward the testicle. In females, it may radiate toward the labium majoris.
- Nausea and vomiting. GI upset may be the only symptom in patients with chronic stones and infection, as seen in xanthogranulomatous pyelonephritis (XGP).
- Fever.
- Abdominal distention from ileus.
- Gross or microhematuria.

EXAM TIP

Typical scenario: A 40-year-old man presents with sudden onset left-sided flank pain that he rates a 10/10. He is writhing, unable to stay still or fnd a comfortable position. Think: Renal colic. Check a urine dip for blood and order a noncontrast CT scan.

diagnost ic t est s

- **Urinalysis and culture:**
  - Some patients will have RBCs in the urine (absence does not rule out stones).
  - White blood cells (WBCs) or bacteria may suggest underlying UTI and should be aggressively treated.
- **Radiographic studies:**
  - Noncontrast abdominal/pelvic CT (Figure 26-3):
    - Fast, requires no IV contrast.
    - Most useful to diagnose small stones (95% sensitivity), hydronephrosis, hydroureter, and perinephric stranding.
    - Useful for revealing other abdominal/pelvic pathology.
    - Study of choice!
  - Plain abdominal film (KUB): Only radiopaque stones ~5 mm will be seen (60–70% specific in the diagnosis of a calculus). However, it is a cheap and quick test to do and is useful in combination with other studies.
  - Renal US: Fast, easy, and relatively inexpensive. Primarily useful in patients who should avoid radiation, such as pregnant women.

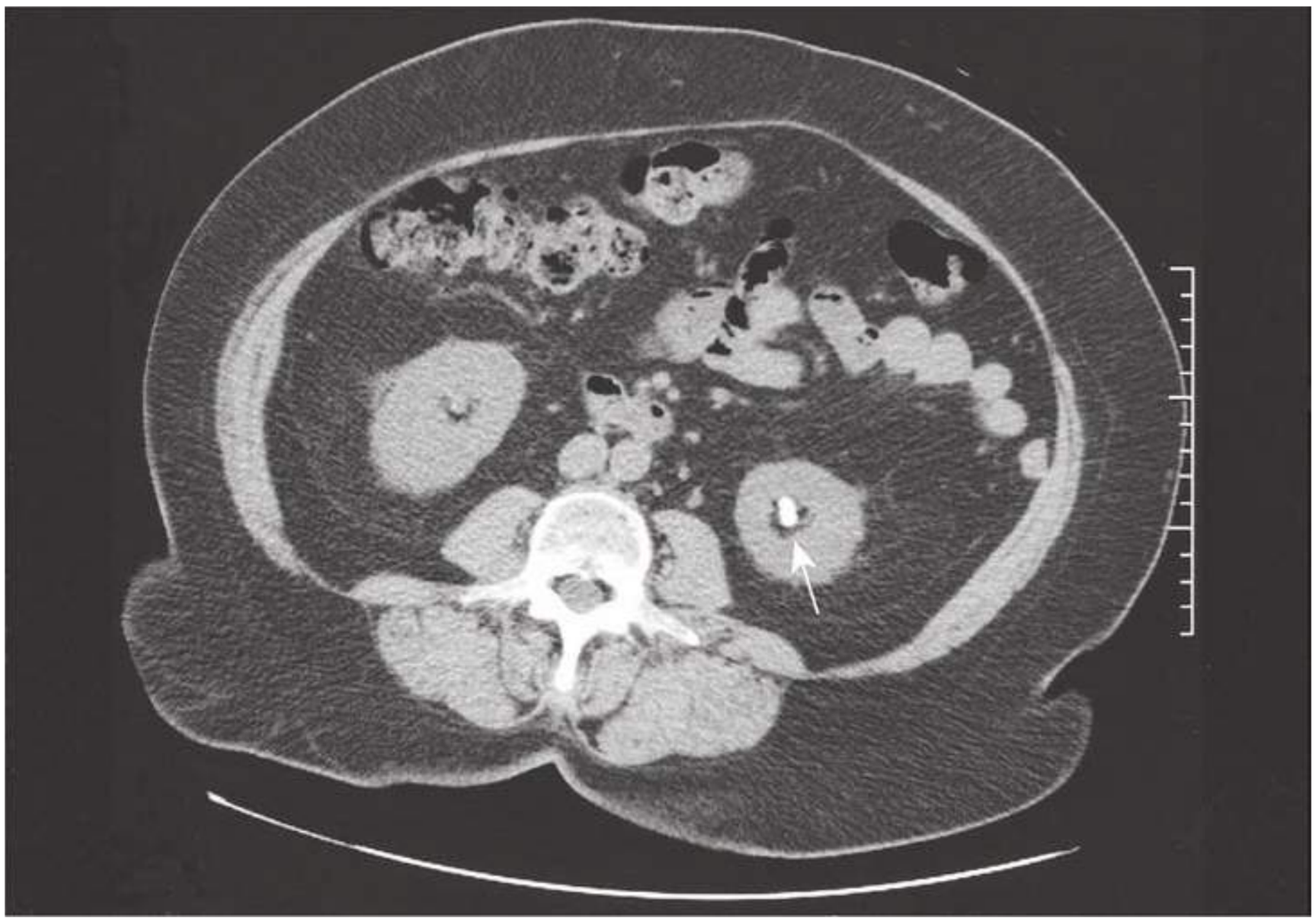


FiGure 26-3. noncontrast abdominal CT demonstrating nephrolithiasis (arrow) in the right kidney, which shows up as radiopaque (white).

management

WARD TIP

Most stones < 5 mm will pass spontaneously in adults.

- Analgesia with nonsteroidal anti-inf ammatory drugs (parenteral ketorolac) and/or opiates.
- IV or PO hydration.
- During passage of a stone, there are five sites where the passage is likely to become arrested. These are narrowest points of the urinary system:
  - Ureteropelvic junction
  - Pelvic brim
  - Iliac crossing
  - Ureterovesical junction
  - Vesicle orifice
- If stone impaction occurs and hydronephrosis develops, decompression of the affected kidney may be necessary to preserve kidney function.
- For stones unlikely to pass spontaneously:
  - Extracorporeal shockwave lithotripsy (ESWL) is effective for stones visible on plain abdominal films (< 2 cm).
  - Ureteroscopy with stone extraction for ureteral calculi and small renal calculi.
  - Percutaneous nephrolithotomy, which establishes a tract from the skin to the collecting system, is used when stones are too large or too hard for lithotripsy.
- Surgical emergency if accompanied by fever, UTI, and/or acute kidney injury, necessitating stent placement or nephrostomy tube.

Benign Prostatic Hyperplasia

definition

- Benign prostatic hyperplasia is the most common benign tumor in men.
- Incidence is directly proportional to age, affecting approximately 90% of men > 80.

- The prostate enlarges (within the confines of a tight prostatic capsule) during puberty when it undergoes androgen-mediated growth. It remains stable in size until about the fifth or sixth decade, when its size increases again.

PathoPhysiology

- Hyperplasia begins in the periurethral area, then progresses to the remainder of the gland; hence, the most common initial symptoms are of urinary outflow obstruction.
- Histologically, the hyperplastic tissue is comprised of glandular epithelium, stroma, and smooth muscle.
- As hyperplasia increases with increasing obstruction, frank urinary retention can occur or may be precipitated by extrinsic etiologies, such as infection, anticholinergic drugs, α-agonists, or alcohol.

signs and symPtoms

- Early symptoms include hesitancy in initiating voiding, weak stream, postvoid dribbling, and the sensation of incomplete emptying.
- As the amount of residual urine increases symptoms may include nocturia, overflow incontinence, and urinary frequency and urgency.

diagnosis

- Clinical history.
- DRE—in hyperplasia, the prostate will be smooth, firm, but enlarged.
- Measurement of postvoid residual urine volume and uroflow.

management

- **Medical:**
  - 5-α reductase inhibitor (finasteride, dutasteride): Blocks conversion of testosterone to dihydrotestosterone, may shrink prostatic hyperplasia up to 20%.
  - α-adrenergic antagonists (prazosin, terazosin, tamsulosin) decrease urethral resistance.
- **Surgical** (it is important to note that these procedures are not cancer surgeries):
  - Transurethral prostatectomy (TURP) for smaller prostates.
  - Open simple prostatectomy for larger prostates.
  - Retrograde ejaculation is a consequence of these surgeries.

Prostatic Carcinoma

definition

- The most common malignancy in men in the United States.
- Rare before age 50.

riskfactors

- Increasing age
- African-American

EXAM TIP

Typical scenario: A 65-year-old male who complains of frequent urination at night with difficulty starting the urine stream. Once starting the urine stream, he states he has difficulty stopping the stream. Think: Prostatic hyperplasia.

EXAM TIP

Typical scenario: An 87-year-old man with a history of prostate cancer presents with low back pain. Think: Bony metastases and possibly cord compression.

classification

- Ninety-five percent are adenocarcinoma. The remaining 5% are squamous, transitional cell, sarcoma, and occasional metastatic tumors.
- Predilection to originate in the peripheral zone and is often multifocal.

signs and symPtoms

- Most patients are asymptomatic at the time of diagnosis, secondary to widespread use of PSA screening tests. It is important to note, that patients on 5-a reductase inhibitors have artificially reduced PSA levels (up to 50%).
- In symptomatic patients, common symptoms include obstructive or irritative voiding complaints.
- Metastatic disease to the bone may cause bone pain.
- Symptoms in advanced disease may include ureteral obstruction, spinal cord compression, deep venous thrombosis (DVT), and pulmonary emboli.

diagnosis

- On DRE, the prostate is usually hard, nodular, fixed, and irregular.
- Prostate-specific antigen (PSA) is the most sensitive test for early detection of prostatic cancer. Following diagnosis, PSA is used to follow progression of disease and response to treatment. However, the PSA is not a specific test. PSA can be elevated in prostatic hyperplasia or prostatitis. Although debate is ongoing, a PSA of 4 in a male over the age of 50 is generally agreed upon as indication for transrectal ultrasonography (TRUS) biopsy.
- **Imaging studies:**
  - TRUS, used for image-guided biopsies: Carcinomas appear as hypoechoic densities in the peripheral zone, has very low sensitivity and specificity.
  - MRI or CT may also be helpful in identifying metastasis and lymph node involvement.
- **Biopsy:** Essential in establishing the diagnosis of prostate cancer.

WARD TIP

Ten to 15% of prostate cancer cases may have normal PSA, hence the importance of the DRE.

staging

- Metastatic spread occurs via:
  - Direct extension (into the seminal vesicles and/or bladder f oor).
  - Lymphatic spread to obturator, internal iliac, common iliac, presacral, and periaortic nodes.
  - Hematogenous spread occurs to bone more frequently than viscera.
- Standard staging scheme used is the **tumor-node-metastases (TNM) system.**
- Gleason grading system is based on a histologic evaluation of prostate tissue samples. The Gleason score is the sum of the two most common cell patterns seen in the tissue sample. The patterns can range from 1 (well differentiated) to 5 (poorly differentiated, highly malignant). Overall scores range from 2 to 10. A grade of 2 has the best prognosis, while a grade of 10 represents poorly differentiated tissue and confers the worst prognosis.
- Bony metastasis can contain both osteoblastic components. Axial skeleton is most commonly affected. Skeletal survey has a low sensitivity for detecting bony metastasis. Radionuclide bone scan has a much higher sensitivity and is also useful in monitoring progression and response to therapy.
- CT of abdomen and pelvis is used to assess lymph node involvement.

treatment

- Treatments vary in aggressiveness depending on patient age, health status, and stage/grade of disease. Treatments may include any/all of the following:
  - Watchful waiting/active surveillance.
  - Androgen deprivation therapy: It has been established that prostatic carcinomas are hormonally dependent. Some degree of control can be obtained by hormonal therapy. Androgen deprivation can be achieved via:
    - Surgical castration (bilateral orchiectomy results in 90% reduction in testosterone).
    - Gonadotropin-releasing hormone (GnRH) agonist (leuprolide) therapy.
    - Androgen receptor antagonists (flutamide, bicalutamide) and/or androgen synthesis inhibitors (ketoconazole).
    - Estrogen administration.
  - Radiation: Can be via XRT or brachytherapy (radioactive seed placement).
  - Radical prostatectomy: May be retropubic (transabdominal), transperineal, laparoscopic, or robotic.
  - Chemotherapy is not very effective, but may be used as a last resort in cases of very advanced, hormone-refractory disease.
  - Combinations of all of the above are commonly used.

WARD TIP

Complications of radiation therapy for prostate cancer:

- Cystitis
- Acute proctitis (diarrhea)
- Urethritis (can lead to strictures)
- Rectal strictures and fstula
- Impotence
- Secondary malignancy

Renal Cell Carcinoma (RCC)

etiology

- Eighty-five percent of all primary malignant renal neoplasms.
- Peak incidence between 50 and 60 years.
- Male-to-female ratio: 2:1.
- Environmental factors: Cigarette smoking; exposure to cadmium, asbestos, and solvents.
- Hereditary link: Genetic defect linked to translocations between chromosomes 3 and 8.

signs and symPtoms

- Classic triad of gross hematuria, fank pain, and palpable abdominal mass (seen in 10–15% of cases).
- Most commonly an incidental finding.
- Most often diagnosed via its systemic symptoms: Fatigability, weight loss and cachexia, intermittent fever, and anemia.
- Other symptoms may relate to the production of hormones and hormone-like substances (paraneoplastic syndromes):
  - Hypercalcemia (parathyroid hormone)
  - Galactorrhea (prolactin)
  - Cushing's syndrome (glucocorticoid)

diagnosis

- It is most important to differentiate cystic from solid lesions.
- Ultrasound has improved the ability to differentiate a solid from a cystic lesion.
- CT is the method of choice for diagnosis and staging of RCC.

staging

- **Stage I:** Tumor confined within the kidney parenchyma.
- **Stage II:** Invasion through the kidney capsule, involves perinephric fat but confined within Gerota's fascia.
- **Stage III:** Involvement of regional lymph nodes, ipsilateral renal vein, or vena cava.
- **Stage IV:** Distant metastasis, adjacent organ involvement.

treatment

- Radical nephrectomy is the treatment of choice if there is no evidence of metastasis. Partial nephrectomy (complete resection of mass) is oncologically equal to radical nephrectomy.
- Radiation therapy can be used in the palliation of patients with metastatic RCC.
- There are no standard chemotherapeutic regimens or hormonal therapy for metastatic disease, and these treatments have been employed with limited success.
- Chemotherapeutic agents (sorafenib).

Prognosis

Five-year survival rates:

Stage I: 91–100%
Stage II: 74–96%
Stage III: 59–70%
Stage IV: 16–32%

Urothelial Cancer

definition and epidemiology

The lining of the urinary system from the renal pelvis to the urethra is made up of transitional cells. This entire lining is subject to carcinomatous changes. However, the bladder is involved most frequently and will therefore be discussed most extensively.

Transitional Cell Carcinoma of the Collecting System and Ureters

- Collecting system and ureteral lesions have a high (~60%) likelihood of spreading to the bladder, as this disease is a field defect. Rate of contralateral spread is ~5%, so patients require lifelong surveillance.
- Nephroureterectomy is treatment of choice.
- As with bladder cancer (see below), advanced disease has poor prognosis.
- Presentation and risk factors are similar to bladder (see below).

Bladder Cancer

- Second most common cancer of the GU tract.
- Men are affected three times more than women.

- Peak incidence occurs between the ages of 60 and 70.
- Ninety-eight percent of bladder cancers are epithelial; in the United States, most are transitional cell carcinomas.
- Nontransitional cell carcinomas, such as squamous cell and adenocarcinomas have a worse prognosis compared to transitional cell carcinoma.
- Squamous cell carcinoma (SCC) tends to arise secondary to chronic irritation and inf ammation (indwelling catheter, recurrent UTI, schistosomiasis).
- Adenocarcinoma tends to be metastatic or direct spread from adjacent organ (large bowel, uterus, uracus).
- Transitional cell carcinoma (by far the most common!) may result from cigarette smoking (twofold increased risk), dye, or chemical exposures.

signs and symPtoms

- Gross and microscopic hematuria are the most common presenting symptoms (80–95%).
- Other symptoms include dysuria, urinary frequency, urgency, and ureteral obstruction.

diagnosis and staging

- Urine cytology for exfoliated malignant cells (poor sensitivity, high specificity).
- Intravenous pyelography (IVP)—ureteral obstruction with hydronephrosis or filling defect.
- Cystoscopy with tumor biopsy.
- Additional staging may be obtained via CT of abdomen and pelvis and endoscopic resection of a bladder neoplasm.
- **Superficial:**
  - Tis: Carcinoma in situ, mucosal involvement.
  - Ta: No invasion.
- **Invasive:**
  - T1: Submucosal involvement.
  - T2: Involvement of bladder muscularis.
  - T3: Involvement of perivesical fat.
  - T4: Involvement of adjacent viscera.
- **Metastatic:** Uses TNM system.

t r eat ment

- Transurethral resection of bladder tumor (TURBT): Endoscopic resection is necessary for initial evaluation of the lesion to assess for depth of invasion. Superficial localized tumors can be treated with TURBT and surveillance (Tis–T1).
- Intravesical chemotherapy using BCG (bacillus Calmette–Guérin) or mitomycin has been shown to have preventative effects after TURBT for lesions up to and including T1.
- Radical cystectomy is the treatment of choice in patients with T2 and greater disease who can tolerate a surgery of this magnitude. Resection of the iliac lymph nodes during this surgery has shown to be both diagnostic and therapeutic.
- The oncologic indications for partial cystectomy are the same as for radical cystectomy. However, the former is generally performed when the tumor(s) are in a location in the bladder, and of a sufficiently small size, amenable to a bladder-sparing procedure. Lymph node dissection is also performed with this surgery.

- Neoadjuvant chemotherapy before (partial) cystectomy improves survival.
- XRT is a reasonable option for muscle invasive (T2–T3) lesions for poor surgical candidates. There is less long-term data on this modality.
- The prognosis of metastatic urothelial cancer is poor. However, chemotherapeutic agents have been shown to prolong survival. Commonly used agents are cisplatin, methotrexate, gemcitabine, doxorubicin, cyclophosphamide, and vinblastine.

Prognosis

The survival rate of patients with metastatic disease is generally < 2 years.

HIGH-YIELD FACTS IN

Orthopedics

Fractures 430
- Open Fracture 430
- pathological Fracture 430
- Stress or Fatigue Fracture 430
- comminuted Fracture 430
- greenstick Fracture 431

Fat Embolism Syndrome 431
- definition 431
- common causes 432
- pathophysiology 432
- Signs and Symptoms 432
- diagnosis 432
- treatment 432
- prognosis 432

Shoulder Dislocations 432
- complications common to all dislocations 433
- complications Specific to type of dislocation 433
- radiograph 434
- treatment 434

Compartment Syndrome 434
- definition 434
- causes 434
- Signs and Symptoms 434
- diagnosis 435
- treatment 435

Osteomyelitis 435
- acute 435
- chronic 436

Brodie's Abscess 437

Septic Bursitis 437

Low Back Pain 437
- epidemiology 437
- history 438
- physical examination 438
- diagnosis 438
- treatment 438

Bone Tumors 438
- radiography 439
- Benign Bone tumors 439
- malignant tumors 441
- metastatic tumors 444

Fractures

- Categorized by anatomical location (proximal, middle, or distal third of shaft), direction of fracture line (transverse, oblique, spiral), and whether it's simple or comminuted.
- Patient presents with loss of function, severe pain, tenderness, swelling, abnormal motion, and often deformity.

Open Fracture

- Fracture communicates with the external environment due to a breach of the overlying soft tissue.
- **True orthopedic emergency:** Almost always results in bacterial contamination of soft tissues and bone.
- Prognosis dependent on extent of soft tissue injury and by type/level of bacterial contamination.
- **Treatment plan:** Prevent infection with early antibiotics and tetanus, debride affected soft tissues and bone, restore available soft tissue, achieve bony union, and institute early joint motion and muscle rehabilitation.

WARD TIP

Osteoporosis is the most common pathologic condition associated with pathologic fractures.

pathOlOgic Fracture

- Occurs due to minimal trauma on a bone weakened by preexisting disease.
- **Predisposing conditions:** Primary or metastatic carcinoma, osteoporosis, bone cysts, enchondroma, giant cell tumors, osteomalacia, osteogenesis imperfecta, scurvy, rickets, and Paget's disease.
- Orthopedic surgeon must not only treat the broken bone but should also diagnose and treat the underlying condition.

WARD TIP

Suspect violence or battering if fracture occurs on normal bone and history reveals trivial trauma.

StreSS Or Fatigue Fracture

- An incomplete or complete fracture resulting from repetitive application of minor trauma.
- Most stress fractures occur in the lower extremities and commonly affect individuals involved in sports and military recruits ("march fracture").
- Pathophysiology of stress fractures is unclear but possibly due to inability of the fatigued muscle to protect bone from strain.
- If the patient is seen within first 2 weeks of onset of symptoms, the plain radiograph is likely to be normal.
- Patients usually complain of pain only with activity.
- **Treatment:** Decrease physical activity or period of limited weight bearing.

WARD TIP

Incidence of stress fractures by site:
Metatarsals: > 50% stress fractures
Calcaneus: 25%
Tibia: 20%
Tarsal navicular: Basketball players

cOmminuted Fracture

Fracture in which the bone is divided into more than two fragments by fracture lines.

salter–harris Fracture

- Fracture involving the physis (growth plate). Occurs irregularly through the weak zone of hypertrophic cartilage (Figure 27-1).
- **Type I:** Fracture is transverse and does not travel vertically across the germinal cell layer. Prognosis for normal healing is good.
- **Type II:** Fracture extends from physis into metaphysis of bone.

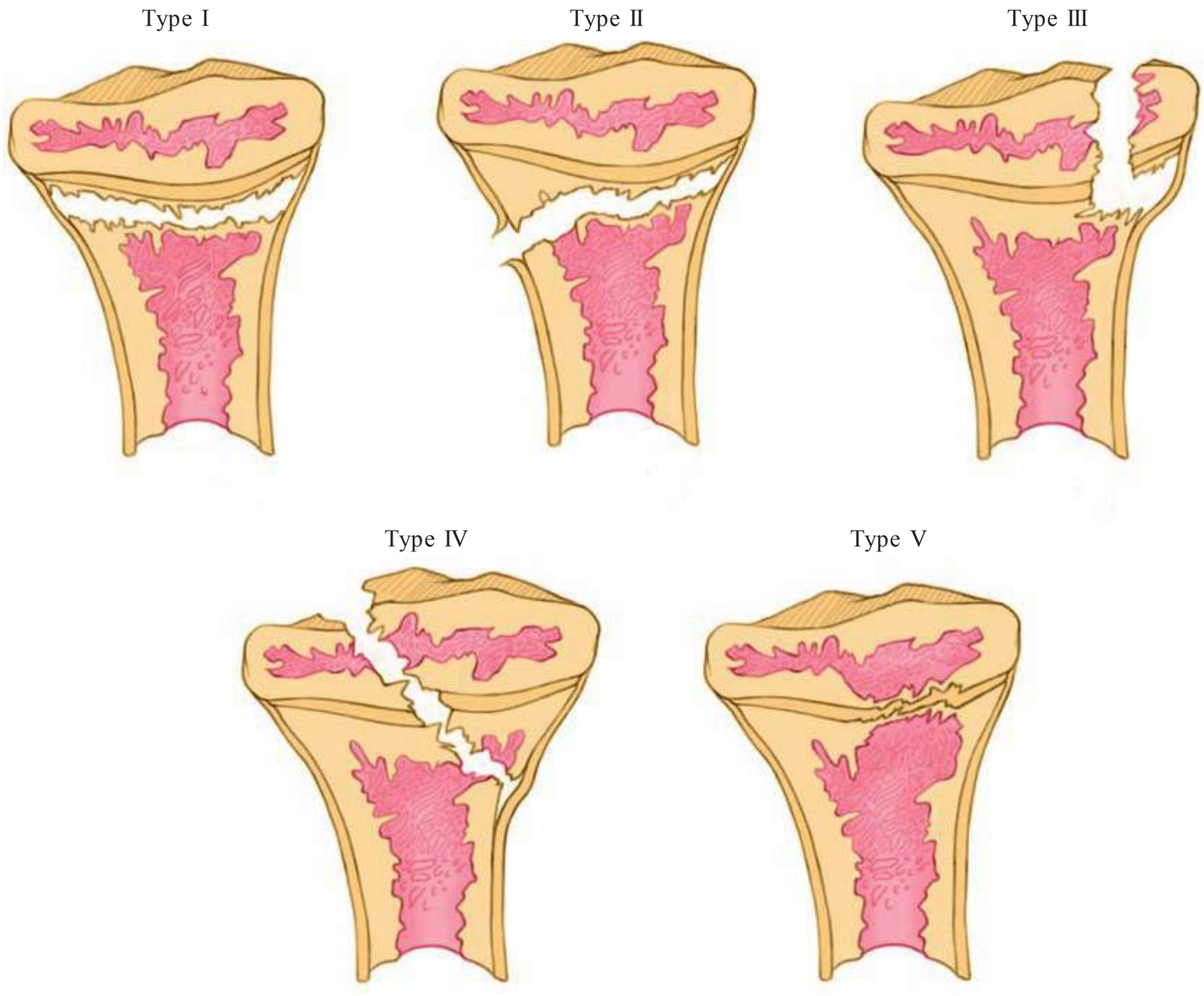


FiGUre 27-1. salter–harris classification of fractures.

- **Type III:** Fracture extends from physis to epiphysis.
- **Type IV:** Fracture traverses the growth plate in a vertical fashion often causing angular deformity from continued growth. Surgical intervention may be necessary.
- **Type V:** Crush injury to the physis such that metaphysis and epiphysis are impacted on one another. No visible fracture line. Poor prognosis with high risk of growth arrest.

WARD TIP

Greenstick fracture: Think of a young, moist twig, which would break without snapping apart!

greenStick Fracture

- An incomplete and angulated fracture of the long bones. A transverse crack that hangs on to its connection.
- Very common in children, rarely seen in adults.
- Since kids have "softer," less brittle bone and thicker (leathery) periosteal membrane, they get incomplete fractures with unique patterns.

Fat Embolism Syndrome

deFiniTiOn

An acute respiratory distress syndrome caused by release of fat droplets from the marrow as may occur following a long bone fracture.

EXAM TIP

Classic triad for fat embolism:
- Confusion
- Dyspnea
- Petechiae on chest

EXAM TIP

typical scenario: A 25-year-old male complains of difficulty breathing. His family notes he is acting a little confused, and that he has a spotty purplish rash. Two days ago, he sustained a femur fracture after a high-speed motor vehicle collision. Think: Fat embolism syndrome.

Common Causes

- Long bone fracture.
- Use of intramedullary nails for treatment of fractures.
- Burns.
- Severe infection.
- Inhalation anesthesia.
- Metabolic disorders.
- Cardiopulmonary bypass.
- Decompression sickness.
- Others: Hemoglobinopathy, collagen disease, diabetes, renal homotransplantations.

Pathophysiology

- Microdroplets of fat are released into the circulation at the site of fracture, occluding pulmonary circulation causing ischemic and hemorrhagic changes.
- Another theory: Release of free fatty acids from the marrow has toxic effects in all tissues, especially the lung.

Signs and Symptoms

- Symptoms may occur immediately or 2–3 days after trauma.
- Shortness of breath with respiratory rate above 30.
- Confusion, restlessness, disorientation, stupor, or coma.
- Fleeting petechial rash on chest, axilla, neck, and conjunctiva.
- Fever, tachycardia.

Diagnosis

- **Hallmark finding:** Arterial hypoxemia. Arterial $PO_2 < 60$ mmHg is suggestive.
- Chest x-ray (CXR): Progressive snowstorm-like infiltration.
- Cryostat—frozen section of clotted blood reveals presence of fat.
- Absence of fat globules in urine makes diagnosis unlikely; however, their presence is not specific for fat embolism.

Treatment

- Administer oxygen to decrease hypoxemia and monitor PO_2 to maintain it over 90 mmHg.
- In severe hypoxemia: Mechanical ventilatory support.
- Use of ethanol, heparin, hypertonic glucose, or steroids has been suggested but their effectiveness is questionable.
- Prevent fat embolism syndrome by careful, early appropriate stabilization of fractures and effective treatment of shock.

Prognosis

Mortality from fat embolism thought to be as high as 50% following multiple fractures.

Shoulder Dislocations

See Table 27-1.

TABLE 27-1. Shoulder dislocations

	Anterior Dislocation	Posterior Dislocation	Inferior Dislocation (Luxatio erecta)
Features	High risk of recurrence 70% Occur in patients younger than 30 years of age	Diagnosis missed in 60% of cases Often precipitated by a convulsion, seizure, electrical shock, and falls	< 1% of all shoulder dislocations
Types	Subcoracoid (most common), subclavicular, subglenoid	Subacromial (most common), subglenoid, subspinous	
Mechanism of injury	Abduction and external rotation of the arm causes strain on anterior capsule and glenohumeral ligaments	Internal rotation and adduction (when one falls on an arm that is forwardly flexed and internally rotated)	Hyperabduction always results in detachment of rotator cuf
Signs and symptoms	▪ Arms held to the side ▪ Patient resists medial rotation and adduction ▪ Prominent acromion ▪ Loss of normal rounded shoulder contour	▪ Patient holds arm medially rotated and to the side ▪ Abduction limited ▪ External rotation limited ▪ Prominence of the coracoid process and posterior part of shoulder ▪ Flattening of anterior aspect of shoulder	▪ Patient in severe pain ▪ Arm held in 180° elevation ▪ Arm appears shorter compared to opposite side ▪ Humeral head often felt along the lateral chest wall

Complications common to all dislocations

Axillary artery injury (more common with luxatio erecta), venous injury, injury to nerves of brachial plexus (most common being axillary nerve):

- Palpate radial pulse to check axillary artery.
- Check motor component of axillary nerve by assessing strength of the deltoid muscle.
- Check sensory component of axillary nerve by assessing sensation over the lateral part of upper arm.
- Do a neurologic exam to evaluate all brachial nerve lesions.

Complications specific to type of dislocation

Anterior Dislocation	Posterior Dislocation	Inferior Dislocation
▪ Rotator cuf tear ▪ Glenoid labral lesions ▪ Coracoid fractures ▪ Greater tuberosity fractures (seems to decrease recurrence) ▪ Hill–Sachs deformity (compression fracture of the posterolateral humeral head)	▪ Fractures of the lesser tuberosity ▪ Fractures of posterior glenoid rim and proximal humerus humeral head)	▪ Rotator cuf tear ▪ Fractures of greater tuberosity

Radiograph

Anterior Dislocation	Posterior Dislocation	Inferior Dislocation
▪ Obtain anterior, posterior, and axillary views ▪ Look for Hill–Sachs deformity in the posterolateral portion of humeral head (occurs in 50%)	▪ Look for loss of the normal elliptical pattern produced by the overlap of humeral head and posterior glenoid rim ▪ Look for the greater tuberosity rotated internally	▪ Top of humerus is displaced downward

Treatment

Anterior Dislocation	Posterior Dislocation	Inferior Dislocation
▪ Reduction (Hennipen technique, Stimson technique, traction and countertraction and lateral traction) ▪ Immobilize shoulder ▪ Surgery if needed	▪ Apply longitudinal traction (Stimson technique) ▪ Surgery if needed, although neurovascular complications are rare	▪ Rotate arm inferiorly while applying traction longitudinally along the humerus with countertraction in the supraclavicular region ▪ Surgical repair of rotator cuf

Compartment Syndrome

Definition

A condition in which increased pressure within a limited space compromises the circulation and function of tissues within that closed space.

Causes

- Fractures.
- Soft tissue crush injuries.
- Vascular injuries.
- Drug overdose with prolonged limb compression.
- Burn injuries.
- Trauma.
- Muscle hypertrophy and nephrotic syndrome.

Signs and Symptoms

- Clinical presentation often indefinite and confusing.
- Hallmark finding: PAIN! Pain in a conscious and fully oriented person that is out of proportion to injury or findings. Must always consider

WARD TIP

do not delay treatment of compartment syndrome! Elevation of pressure to > 30 mmHg for > 8 hours leads to irreversible tissue death.

compartment syndrome if pain not controlled with appropriate treatment. Can also see paresthesia at the first web space (i.e., between great and second toes) but this is often later and concerning for permanent injury.

- **P**ain: Deep, unremitting, and poorly localized. Pain increases with passive stretching of involved muscle.
- **P**allor: Not necessary for diagnosis, may not be present.
- **P**aresthesias: Of cutaneous distribution supplied by the compressed nerve is an early sign.
- **P**aralysis: Occurs after ischemia is well established.
- **P**ulselessness: Shown to occur late at times. Pulse may be present.
- Compartment tense on palpation.

diagnosis

This is a clinical diagnosis. Diagnosis made with recognition of high-risk patients and uncontrollable pain out of proportion with injury. Be aware of distracting injuries. Measure pressure within compartment with commercially available monitors.

- Pressure < 30 mmHg usually will not produce a compartment syndrome.
- Pressure > 30 mmHg is an indication for fasciotomy.

treatment

Complete fasciotomy: Goal is to decompress all tight compartments and salvage a viable extremity.

Osteomyelitis

acute

pathophysiology

- Bacteria lodge in end artery of metaphysis and multiply.
- Local increase in serum and white blood cells (WBCs).
- Decrease in blood flow and pressure necrosis.
- Pus moves to Haversian and medullary canals.
- Goes beneath the periosteum.

WARD TIP

Most common site for acute osteomyelitis is the metaphyseal end of a single long bone (especially around the knee).

causes

- Route of infection is mainly hematogenous, rarely trauma.
- Most cases of acute hematogenous osteomyelitis caused by Staphylococcus aureus.

signs and symptoms

- History of infection (e.g., skin or throat) or trauma.
- Significant pain in the affected area, anorexia, fever, irritability, nausea, malaise, rapid pulse.
- Limited joint motion, tenderness, swelling of soft tissue, inability to bear weight and guarding apparent on physical exam.

diagnosis

- Elevated WBCs, erythrocyte sedimentation rate (ESR), and C-reactive protein; ± anemia.
- Deep circumferential soft-tissue swelling with obliteration of muscular planes.

differential diagnosis

- Septic arthritis: Swelling and tenderness directly on the joint with intense pain on joint movement, high WBC, and positive culture.
- Rheumatic fever: More insidious onset, less local and constitutional symptoms.
- Ewing's sarcoma: Early symptoms are more insidious and less intense and can present with bone destruction.

treatment

- **Medical:** Infection must be diagnosed early. Intravenous antibiotics (usually oxacillin or cloxacillin 8–16 g adult) started soon after obtaining specimen for culture. Monitor temperature, swelling, pain, WBC, and joint mobility.
- **Surgical:** Open drainage of abscess if antibiotics fail or signs of abscess appear. After surgical drainage, wound is left open to heal by secondary intention.

chronic

epidemiology

Often seen in lower extremities of a diabetic patient.

pathophysiology

- Untreated acute osteomyelitis results in a cavity walled off by an involucrum containing granulation tissue, sequestrum, and bacteria.
- Drainage of pus into surrounding soft tissue and skin via sinus tracts.
- Persistent drainage can lead to carcinoma.
- Bone fragments and exudates unreachable by antibiotics.
- Result is severely deformed bone and possibility of pathologic fracture.

causes

- Usually an end result of untreated or treatment failed acute osteomyelitis. Occasionally due to trauma or surgery.
- Cause is usually polymicrobial—difficult to eradicate.

signs and symptoms

- Characterized by persistent drainage following an episode of acute osteomyelitis or onset of inflammation and cellulitis following an open fracture.
- Fever (not always present), pain, mild systemic symptoms, tenderness.
- Easy to diagnose when drainage is present and x-ray shows bone destruction and deformity. In cases with absence of drainage, radionuclide imaging studies can be helpful.

diagnosis

Radiographic findings:

- Areas of radiolucency within an irregular sclerotic bone.
- Irregular areas of destruction present. Often, periosteal thickening can be seen.

differential diagnosis

- Acute suppurative arthritis.
- Rheumatic fever: Examine synovial fluid.
- Cellulitis: Absence of soft tissue swelling on radiographs.

treatment

- Varies from open drainage of abscess or sequestrectomy to amputation.
- Most effective: Extensive debridement of all necrotic and granulation tissue along with reconstruction of bone and soft tissue defects with concomitant antibiotics.
- Excellent adjunct: Temporary placement of absorbable or nonabsorbable antibiotic containing beads in the wound for local administration of antibiotics.

complications

- Soft tissue abscess.
- Septic arthritis due to extension to adjacent joint.
- Metastatic infections to other areas.
- Pathologic fractures.
- If significant spinal involvement, paraplegia.

Brodie's Abscess

- Subacute pyogenic osteomyelitis in the metaphysis.
- X-ray finding: Lucent lesions surrounded by sclerotic bone.
- Usually caused by S. aureus and Staphylococcus albus.

Septic Bursitis

- Infection of the superficial bursa commonly affecting the bunion, olecranon, and prepatellar bursa.
- Most common offending organism is S. aureus.
- Clinically presents with painful bursal swelling, often along with intense cellulitis. Systemic signs of sepsis can be present along with regional lymphadenopathy.
- Treatment: Aspirate bursa for culture and sensitivity. Give broad-spectrum antibiotic. Take care not to aspirate the joint since passing the needle through the area of cellulitis might spread it to the joint! Can require surgical debridement or bursectomy.

Low Back Pain

epidemiology

- Four out of five people suffer from low back pain sometime in life.
- Incidence 15–20%, males > females.
- Most patients with low back pain have no systemic disorder.
- Back pain can be symptom of a systemic illness such as primary or metastatic neoplasm, infectious disease, aortic aneurysm, renal disease or stones, or an inflammatory disorder, especially if very intense or acute onset without antecedent trauma.



WARD TIP

Low back pain is the leading cause of an orthopedic visit.

history

- Very important, although often the only presenting complaint is pain that is poorly localized.
- Character of pain needs to be described: What is the pain like? Does it radiate? When does it occur? How does it interfere with sitting, standing, walking? What factors make the pain better or worse? How many episodes have you had? Any other symptoms along with back pain?
- Give patient a diagram and ask patient to mark areas of pain.
- History of pain development and how it affects everyday life.
- History of weight loss, malaise, fever, gastrointestinal (GI) or genitourinary (GU) illnesses.
- Psychological assessment in patients with chronic pain.

physical examination

- Straight leg-raising test: Positive (reproduces the complaining symptom) in nerve root irritation.
- Check for ref exes and motor and sensory deficits.
- Check presence of nonorganic signs (Waddell's signs) when patient responds to axial loading, local touch, and simulated rotation—signifies faking or exaggerating symptoms for secondary gain.
- Check spine for range of motion.
- Bowel and bladder symptoms are suggestive of cauda equina syndrome (surgical emergency).
- Leg and buttock pain are suggestive of herniated disk.

diagnosis

- X-rays of lumbar spine especially if patient is > 50 years of age and has history of other medical illnesses or trauma.
- Magnetic resonance imaging (MRI) of the lumbar spine if x-rays are negative: Great for assessing neural tissue.
- Computed tomography (CT) scan if MRI not helpful.
- Technetium bone scan and gallium scan can be done if an infection of the spine is suspected.

treatment

- Rule out a serious pathologic condition.
- Goal is early return to normal activities.
- Patients with acute low back pain should avoid sitting or lifting and use mild analgesics and anti-inf ammatory drugs.
- Physical and occupational therapy programs prove to be helpful.
- Antidepressants often help those with pain persistent for 3 months.
- Other treatment options: Transcutaneous electrical nerve stimulation (TENS), traction, manipulation with radicular signs, biofeedback, acupuncture, trigger point injections, and muscle relaxants.

Bone Tumors

- Occur due to uncontrolled cellular proliferation of a single clone of cells whose regulatory mechanisms are defective.
- Benign tumors are 200 times more likely to occur than malignant ones.

- A careful history and physical is very crucial and will reveal the duration of the mass, onset of pain, other associated symptoms, and the chronological sequence of these symptoms.
- A thorough physical exam consists of evaluation of patient's general health status. The mass should be noted for size, location, consistency, mobility, tenderness, local temperature, and change with position. Note any muscular atrophy.

radiography

- Never diagnose a bone tumor without an x-ray.
- Bone reacts to a benign or malignant tumor by bone production or destruction.
- An x-ray appearance can show bone production, destruction, or both.
- Three patterns of x-ray appearance:
  - **Permeative:** Implies a rapidly spreading intramedullary tumor; tumor replaces marrow and fat.
  - **Moth eaten:** Implies a poorly circumscribed, slow-growing malignant tumor.
  - **Geographic:** Implies a well-circumscribed slow-growing tumor, therefore bone has time to react and results in sclerotic margins.

Benign Bone tumors

Patient is usually asymptomatic, and the x-ray shows a well-defined lesion with sclerotic margins.

Osteoid Osteoma

Signs and Symptoms

- Most common osteoid-forming benign tumor (10%).
- Male-to-female ratio is 2:1 to 3:1, three fourths of cases between ages 5 and 25.
- Most common sites: Diaphysis of long tubular bones, especially the proximal femur.
- Local tenderness and dull aching pain that is localizable, tends to be more severe at night and relieved by nonsteroidal anti-inflammatory drugs (NSAIDs).
- Pain can radiate and mimic other diseases (sciatica if present in vertebra).

Radiography

Localized area of bone sclerosis with a central radiolucent nidus. Little sclerosis seen if it is present in cancellous bone.

histology

Usually measures < 1 cm. Circumscribed, highly vascular nidus made of fibroconnective tissue and woven bone.

treatment and prognosis

Mostly treated symptomatically with aspirin or NSAIDs. If this fails, surgical intervention to remove the nidus or radio frequency ablation (RFA). Prognosis is excellent.

Osteoblastoma

epidemiology

- One percent of benign bone tumors. Larger than osteoid osteomas.
- Male > female, most patients between ages 10 and 35 years.
- Most common sites: Mostly axial skeleton, less common in jaw, hands, and feet. One third to one half of cases are in the vertebral column.

EXAM TIP

Emergency causes of low back pain:
FAc t Oid
Fracture
Abdominal aortic aneurysm
c auda equine syndrome
t umor (cord compression)
Other (osteoarthritis [OA], severe musculoskeletal pain, other neurological syndromes)
infection (e.g., epidural abscess)
d isk herniation/rupture

Signs and Symptoms

Pain is the major complaint. Tenderness and swelling may be present over the lesion.

Radiography

Nonspecific x-ray findings, which may be interpreted as osteoid osteomas, aneurysmal bone cysts, or malignant tumors.

Histology

Histologically similar to osteoid osteoma.

Treatment and Prognosis

- Vigorous curettage of the lesion. Prognosis is generally good except occasionally can become locally aggressive or recur locally if they are not adequately excised.
- Have potential to undergo malignant transformation and metastasize.

WARD TIP

Multiple hereditary osteochondroma is an autosomal dominant disorder in which multiple bones have osteochondromas (1% risk of malignant transformation).

Osteochondroma

- An outgrowth of bone capped by cartilage.
- Most common benign tumor of the bone (45%) with most patients in their first two decades of life.
- Usually solitary. Originates in childhood from growing epiphyseal cartilage plate. Mostly stage I lesions.
- Most common sites: Metaphysis of long bones of extremities; rarely in fat bones, vertebrae, or clavicle.

Signs and Symptoms

May be asymptomatic; patient may complain of pain, mass, or impingement syndromes.

Radiography

Shows a mushroom-like bony prominence.

Histology

Trabecular, cancellous bone continuous with the marrow cavity and covered by hyaline cartilage cap.

Treatment and Prognosis

- Surgical excision if the patient complains of pain or if it enlarges after puberty.
- Rarely undergo malignant transformation to chondrosarcoma (< 1%).

WARD TIP

Ollier's disease: Enchondromas in multiple bones. More likely for malignant transformation. Can result in bowing and shortening of long bones.

Enchondroma

- Neoplasm consisting of mature hyaline cartilage (chondroma).
- A centrally located chondroma. Can be single or multiple.
- Ten percent of benign tumors. Peak incidence in ages 20–50 years.
- Most common sites: Tubular bones of hands and feet.
- Chondromas can arise close to cortex or periosteum (ecchondroma) or in relation with synovium, tendons, or joints (synovial chondroma).
- Stage I or stage II lesions.

Signs and Symptoms

Asymptomatic until a pathologic fracture brings attention to it.

Radiography

Geographic lysis in a well-circumscribed area with spotty calcifications.

Histology

Consists of hyaline cartilage often with active nuclei. Interpretation depends on size, location, and growth.

Treatment and Prognosis

No treatment if patient is asymptomatic. If pathologic fracture occurs, then allow fracture to heal. Perform a simple excision and bone grafting procedure.

Giant cell tumor

- Thought to arise from mesenchymal stromal cells supporting the bone marrow.
- Five to 10% of benign bone tumors. Peak incidence in 30s.
- Female-to-male ratio is 3:2.
- Most common sites: Around the knee (distal femur, proximal tibia), distal radius, and sacrum.
- Mostly stage II or III lesions.

Signs and Symptoms

- Pain, swelling, and local tenderness; often presents with arthritis or joint effusions due to proximity to the joint.
- May also present with a pathologic fracture.

Radiography

- A radiolucent lesion occupying the epiphysis and extending into the metaphysis; asymmetrical with bone destruction.
- Occasional "soap bubble" appearance due to a thin subperiosteal bone shell.

Histology

Abundant mononuclear stromal cells interspersed with a lot of giant cells with numerous nuclei.

Treatment and Prognosis

- Curettage and bone grafting (recurrence rate > 50%).
- Aggressive curettage with adjuvant phenol, hydrogen peroxide, or liquid nitrogen (recurrence rate 10–25%).
- Important to obtain CXR or CT every 6 months for 2–3 years for monitoring.
- Often recurs after incomplete removal.

WARD TIP

Factors that predispose to malignancy:

- Size (> 4.5 cm)
- Location (long and axial bones)
- Growth (active and painful)

malignant tumors

Metastatic tumors are much more common than primary tumors.

Osteosarcoma

- Tumor made of a malignant spindle cell stroma producing osteoid.
- Many subtypes of osteoid forming sarcomas.
- Peak incidence in ages between 10 and 30 years, male > female.
- Most common sites: Around the knee (distal femur, proximal tibia), proximal humerus, rarely mandible.

WARD TIP

Osteosarcoma can occur secondary to Paget's disease.

Signs and Symptoms

- Pain associated with a tender mass.
- Dilated veins may be visible on the skin over the mass.
- Constitutional symptoms may be present.

Radiography

- X-ray shows a poorly defined lesion in the metaphysis with areas of bone destruction and formation.
- Codman's triangle: Due to new bone formation under the corners of the raised periosteum.
- Sun-burst appearance: Occurs when the bone spicules are formed perpendicular to the surface of the bone.

histology

- Spindle-shaped tumors cells with odd, hyperchromatic nuclei showing a high mitotic rate. Giant cells may be present.
- High-dose methotrexate, doxorubicin, cisplatin, and ifosfamide along with surgical intervention.
- Tumors hematogenously metastasize to the lung.
- Surgery plus chemotherapy: Five-year survival is about 60%.
- Better prognosis if the tumor is in a small bone.

chondrosarcoma

- Low-grade malignant tumor that derives from cartilage cells.
- Seven to 12% of primary bone tumors; male > female.
- Peak incidence between ages 30 and 60 years.
- Most common sites: Pelvis, femur, fat bones, proximal humerus, scapula, upper tibia, and fibula.

Signs and Symptoms

Pain and swelling over months or years.

Radiography

- Central chondrosarcomas show well-defined radiolucent areas with small, irregular calcifications to ill-defined areas breaking through the cortex.
- Peripheral chondrosarcomas look like large, lobulated masses hanging from the surface of a long bone with calcification.

histology

Varies from well-differentiated hyaline cartilage with little nuclear atypia to highly anaplastic spindle cell tumor with little cartilaginous differentiation.

treatment and prognosis

- Surgical resection of the tumor.
- Do not respond to radiation or chemotherapy.
- Prognosis better than osteosarcoma since chondrosarcoma grows slowly and metastasizes late.

ewing's sarcoma

- Tumor of small round blue cells arising in the medullary cavity.
- Seven percent of primary bone tumors.
- Ninety percent of cases between ages 5 and 25 years; male > female.
- Most common sites: Diaphysis or metaphysis of long bones, pelvis, and scapula; potential to occur anywhere in the body.

EXAM TIP

Ewing's sarcoma is the most lethal of all bone tumors.

Signs and Symptoms

- Pain that increases with time and is more severe at night.
- Local swelling and a tender mass.
- Malaise, fever, leukocytosis, mild anemia, increased ESR.
- Often mimic subacute osteomyelitis, syphlitic osteoperiostitis, or other tumors.

Radiography

- Shows lytic bone lesions with a permeative pattern.
- Elevations and permeations of the periosteum give rise to lamellated "onion skin" appearance.

Histology

Area contains densely packed small round blue cells containing glycogen with little intercellular stroma arranged in sheets, cords, or nests.

Treatment and Prognosis

- Vincristine, cyclophosphamide, actinomycin D, and adriamycin along with surgery or radiation.
- Advanced metastatic disease: Five-year survival is 30%.
- Surgically resectable lesion treated with drugs and surgery has a 70% chance of 5-year survival.
- Males have worse prognosis.

WARD TIP

Ewing's sarcoma usually occurs after age 5. If patient < 5 years of age, think metastatic neuroblastoma instead.

Multiple Myeloma

- A malignant plasma cell tumor with multiple site involvement.
- Most common primary malignant tumor of the bone (45%).
- Ninety percent of cases in patients over the age of 40 years; male > female.
- Most common sites: Vertebral column, ribs, skull, pelvis, femur, clavicle and scapula; can occur anywhere in the body.

Signs and Symptoms

- Bone pain.
- Weight loss, weakness, neurologic impairment if pathologic fractures in the vertebrae present.
- Pathologic fractures or deformities.
- Susceptibility to infections.
- Amyloidosis.
- Kidney damage due to protein plugging of renal tubules.

Labs

- Increased serum calcium due to bone reabsorption.
- Elevated uric acid due to increased cell turnover.
- Monoclonal gammopathy, Bence-Jones proteinuria, increased ESR, and rouleaux formation.
- Anemia due to marrow suppression.

EXAM TIP

Classic triad for multiple myeloma: **pAM**
"**p**unched out" lytic lesions
Atypical plasma cells
Monoclonal gammopathy

Radiography

Classically shows sharply punched out lesions giving a soap bubble appearance or often shows diffuse demineralization.

Histology

Marrow aspirate shows larger-than-normal plasma cells with many nuclei, a nucleoli, and showing mitotic activity.

EXAM TIP

Two percent of myeloma cases will present with **pOeMs**:
polyneuropathy
Organomegaly
endocrinopathy
M-component spike
skin changes/**s**clerosis of bone

Treatment and Prognosis

- Chemotherapy with melphalan, often with prednisone.
- Bisphosphonates and other bone anti-resorptive agents help in reducing pathologic fractures.
- Untreated cases rarely survive more than 6–12 months.
- Chemotherapy induces remission in about 50–70% of cases.
- Poor prognosis with 90% of patients dying within 2–3 years.

Metastatic Tumors

EXAM TIP

Most likely site of origin for metastatic tumors: **Breast, Lungs, thyroid, Kidney, prostate.**

- Metastatic tumors comprise 95% of all malignant bone tumors, primary bone tumors 5%.
- Spread via direct extension, lymphatics, vascular system, or intraspinal seeding.
- In children: Bone metastasis most likely from a neuroblastoma.
- Most common sites: Vertebral column, ribs, pelvis, upper ends of femur and humerus.

Signs and Symptoms

- Pain (most common symptom).
- Spine involvement: Neurologic symptoms due to pressure on nerve roots or spinal cord.
- Hypercalcemia and anemia.
- Pathologic fractures are common.
- Increased acid phosphatase in prostatic metastasis.

Diagnostic Workup

- Order complete blood count (CBC), ESR, liver and renal panels, alkaline phosphatase, and serum protein electrophoresis.
- CT of chest-abdomen-pelvis, x-ray of the entire effected bone.
- Staging bone scan (more sensitive than x-ray).

Radiography

- Lesions may be multiple or solitary (kidney), well or poorly circumscribed, osteoblastic or osteolytic (majority).
- Osteoblastic: Breast and prostate.

Histology

Generally, primary tumors produce matrix for the tumor stroma, whereas epithelial tumors form clusters in fibrous tissue.

Treatment and Prognosis

- Radiation as palliative treatment along with chemotherapy.
- Surgical intervention aims at relieving pain and prevents pathologic fractures.
- Radioactive iodine for thyroid carcinoma metastasis.
- Tamoxifen for metastatic carcinoma of the breast.
- Bilateral orchiectomy, estrogens, or antiandrogens for metastatic prostate tumors.
- Poor prognosis with average survival time being 19 months after suffering a pathologic fracture.

The hand

Anatomy 446
- Muscles 446
- Bones 446
- nerves 447
- Tendons 449

History 450

Physical Examination 450

Nerve Blocks 451

Infections of the Hand 451
- Felon 451
- Paronychia 452
- SePTic Flexor Tenosynoviis 452

Gamekeeper's Thumb 453
- deFiniTion 453
- eTiology 453
- Signs and SyMPToMs 453
- diagnosis 453
- TreaTMenT 453

Carpal Tunnel Syndrome 454
- deFiniTion 454
- eTiology 454
- risk FacTors 454
- ePideMiology 454
- Signs and SyMPToMs 454
- diagnosis 455
- TreaTMenT 455

Ganglion Cyst 455
- deFiniTion 455
- eTiology 455
- Signs and SyMPToMs 455
- diagnosis 455
- TreaTMenT 455

Mallet Finger 455
- deFiniTion 455
- eTiology 456
- Signs and SyMPToMs 456
- TreaTMenT 456

Trigger Finger 456
- deFiniTion 456
- risk FacTors 457
- Signs and SyMPToMs 457
- TreaTMenT 457

Amputation Injuries 457
- indicaTions For Surgery 457
- relaTive conTraindicaTions To Surgical reconsTrucTion 457
- coMPlicaTions 457
- PreoPeraTive ManageMenT 457

High-Pressure Injection Injuries 458
- Signs and SyMPToMs 458
- diagnosis 458
- TreaTMenT 458

Other Common Hand and Wrist Injuries 458

Anatomy

Muscles

- Intrinsic muscles of the hand have their origin and insertion in the hand. See Table 28-1.
- Extrinsic muscles of the hand have their muscle bellies in the forearm and their tendon insertions in the hand. See Table 28-2.

Bones

- There are 27 bones in the hand: 5 metacarpals, 14 phalanges, and 8 carpals.
- Each finger has one metacarpal.
- Each finger or digit (except the thumb) has three phalanges—proximal, middle, and distal.
- The thumb has only two phalanges—a proximal and a distal phalanx.
- The joints between the metacarpal and the proximal phalanx is the metacarpophalangeal (MCP).

TABLE 28-1. Intrinsic Muscles of the hand

Muscle	Innervation	Function
Thenar Group		
Abductor pollicis brevis	Median	Abduction of thumb
Adductor pollicis brevis	Median	Adduction of thumb
Flexor pollicis brevis	Median	Flexes thumb MCP joint
Opponens pollicis	Ulnar (deep branch)	Opposes—pulls thumb medially and forward across palm
Remainder of hand		
Palmar interossei	Ulnar	▪ Adduct finger toward center of third digit ▪ Flex MCP, extend PIP and DIP
Dorsal interossei	Ulnar	▪ Abduct finger from center of third digit ▪ Flex MCP, extend PIP and DIP
Lumbricals	First and second: Median Third and fourth: Ulnar (deep branch)	Flex MCP, extend PIP and DIP
Palmaris brevis	Ulnar (superficial branch)	Aids with hand grip
hypothenar Group		
Abductor digiti minimi	Ulnar (deep branch)	Abducts little finger
Flexor digiti minimi	Ulnar (deep branch)	Flexes little finger
Opponens digiti minimi	Ulnar (deep branch)	Aids little finger with cupping motion of hand

TABLE 28-2. extrinsic Muscles of the hand

Muscle	Compartment	Innervation	Function
Flexor carpi radialis	Anterior	Median	Flexes and abducts hand at wrist
Palmaris longus	Anterior	Median	Flexes hand
Flexor carpi ulnaris (humeral and ulnar heads)	Anterior	Ulnar	Flexes and abducts hand at wrist
Flexor digitorum superficialis (humeroulnar and radial heads)	Anterior	Median	▪ Flexes middle phalanx ▪ Assists with flexion of proximal phalanx and hand
Flexor digitorum profundis	Anterior	Median and ulnar nerves	▪ Flexes distal phalanx ▪ Assists in flexion of middle and proximal phalanx and wrist
Extensor carpi radialis longus	Lateral	Radial	Extends and abducts hand at wrist
Extensor carpi radialis brevis	Posterior	Radial	Extends and abducts hand at wrist
Extensor digitorum	Posterior	Radial	Extends fingers and hand
Extensor digiti minimi	Posterior	Radial	Extends little finger MCP joint
Extensor carpi ulnaris	Posterior	Radial	Extends and adducts hand at wrist
Abductor pollicis longus	Posterior	Radial	Abducts and extends thumb
Extensor pollicis longus	Posterior	Radial	Extends distal phalanx of thumb
Extensor pollicis brevis	Posterior	Radial	Extends thumb MCP joint
Extensor indici	Posterior	Radial	Extends index finger MCP joint

- The joints between the phalanges are the proximal interphalangeal (PIP) and distal interphalangeal (DIP).
- The thumb has only MCP and DIP joints.
- Carpal bones (see Figure 28-1):
  - **S**caphoid
  - **L**unate
  - **T**riquetrum
  - **P**isiform
  - **T**rapezium
  - **T**rapezoid
  - **C**apitate
  - **H**amate

EXAM TIP

The wrist bones are easily remembered by the saying: **S**ome **L**overs **T**ry **P**ositions **T**hat **T**hey **C**an't **h**andle.

nerves

- **Sensory:**
  - Radial: Sensory to lateral aspect of dorsum of hand and radial 3.5 digits.
  - Median: Sensory to skin on lateral half of palm and palmar aspect of the radial 3.5 digits.
  - Ulnar: Sensory to skin on ulnar aspect of dorsum of hand, hypothenar eminence, and ulnar 1.5 digits.
- **Motor:** See Tables 28-1 and 28-2 for muscles innervated by the radial, median, and ulnar nerves.

WARD TIP

The radial nerve does not innervate any of the intrinsic muscles of the hand. See Table 28-3 for clinical maneuvers to test function of the hand.

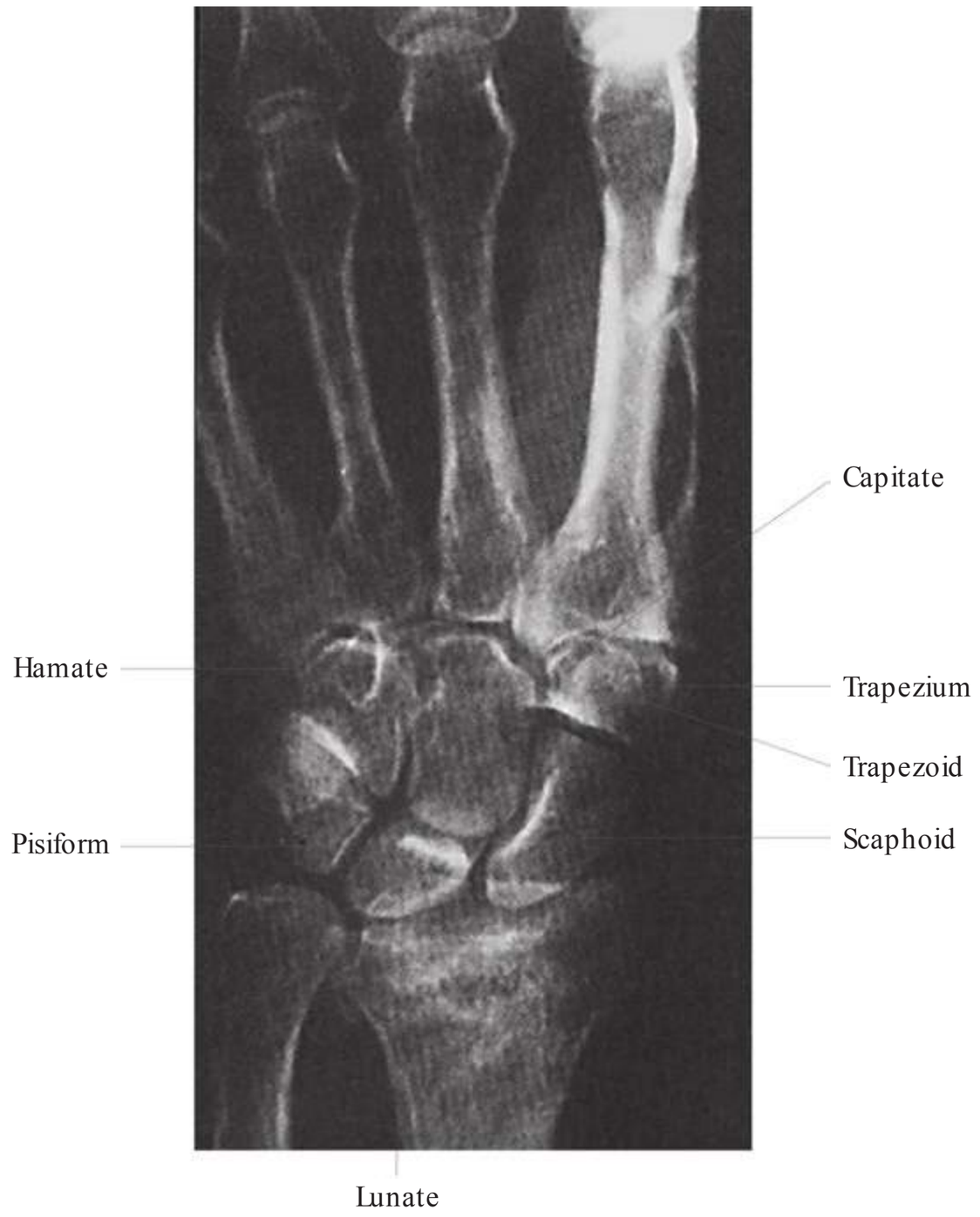


FIGURE 28-1. Bones of the wrist on a P radiograph.

TABLE 28-3. Clinical Maneuvers for Testing Muscles of the hand

Patient Maneuver	Muscle tested
Making a fist	
Bending the tip of the thumb	Flexor pollicis longus
Bending each individual fingertip against resistance while PIPs are stabilized by examiner	Flexor digitorum profundus
Bending each individual fingertip against resistance while DIPs are stabilized by examiner	Flexor digitorum superficialis
Bring thumb out to side and back	Extensor pollicis brevis and abductor pollicis longus
Flexing and extending a fist at the wrist	Extensor carpi radialis longus and brevis
Raising thumb only while rest of hand is laid f at	Extensor pollicis longus
Making a fist with little finger extended alone	Extensor digiti minimi

Tendons

Zones of the hand

- Two main groups: Flexors and extensors.
- Extensor tendon lacerations can usually be repaired in the emergency department (ED).
- Flexor tendons are more difficult and usually require operative repair and careful rehabilitation.
- The f exor and extensor tendons are grouped into zones (see Figures 28-2 and 28-3).
- Flexor tendon injury repair timetable by zone:
  - Zones I and II: 1–3 weeks.
  - Zones III–V: Immediate.
  - Zone IV injuries are technically difficult because tendons lie within the carpal tunnel.
  - Zone V tendon injuries are relatively easy to fix, but functional outcome is often poor due to associated nerve injury.

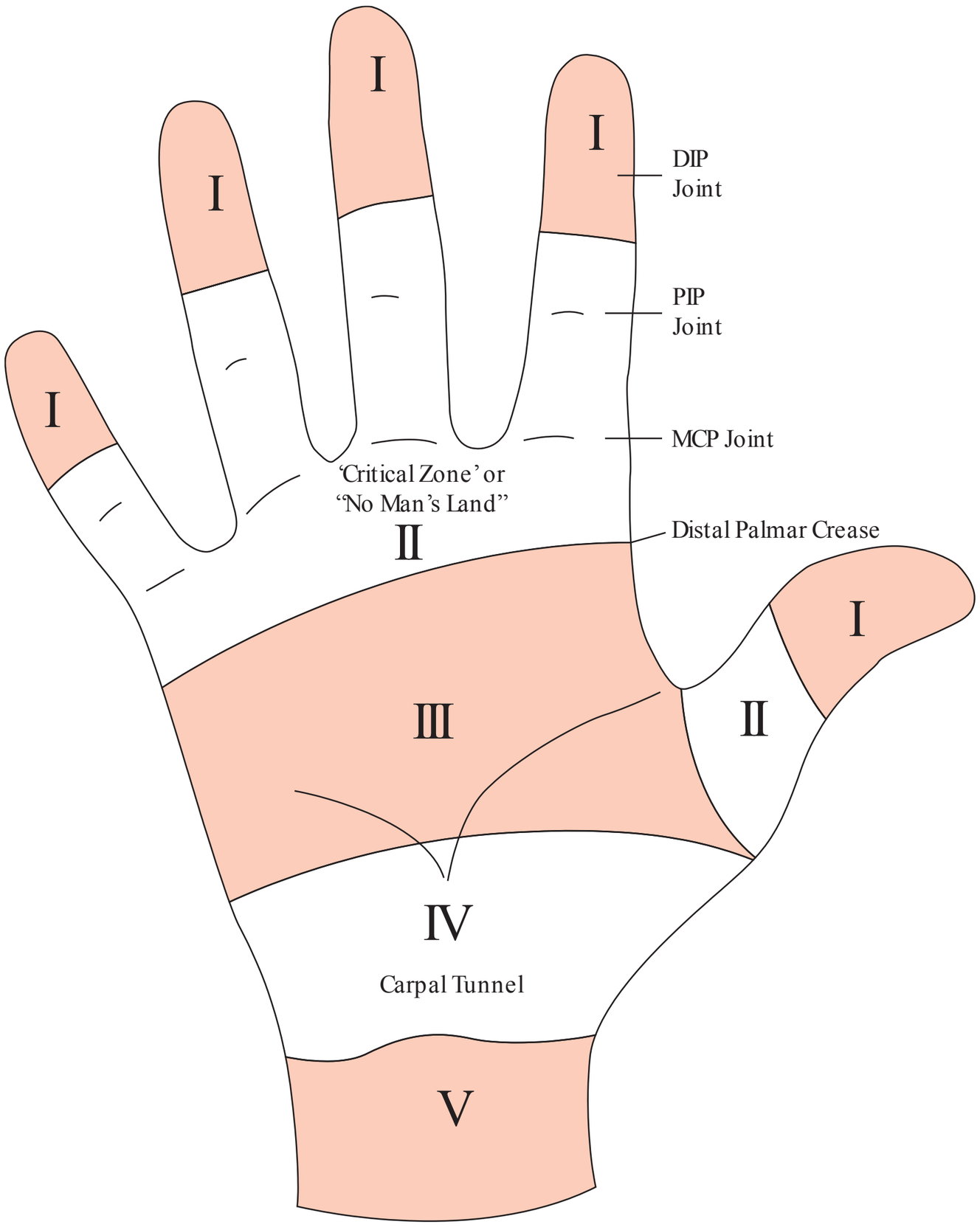


FIGURe 28-2. Flexor tendon zones of the hand. (Artwork by Elizabeth N. Jacobson, Mayo Medical School.)

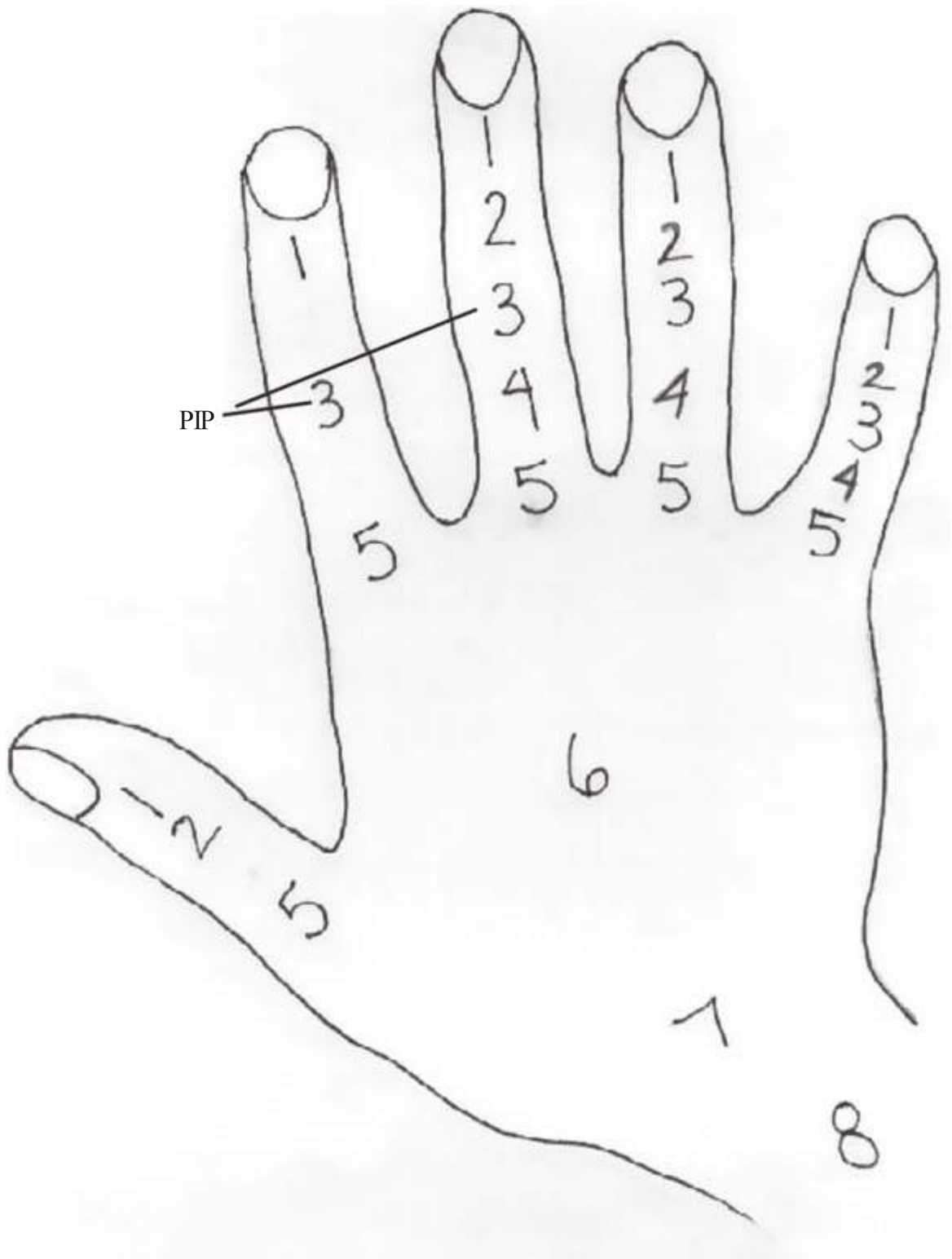


FIGURE 28-3. extensor tendon zones of the hand. (Artwork by Elizabeth N. Jacobson, Mayo Medical School.)

History

Focused hand history:

- Hand dominance.
- Time of injury.
- Status of tetanus immunization.
- Occupation.
- Cause and mechanism of injury.

WARD TIP

A flexor tendon injury will present with a straight finger (unopposed extensors).

Physical Examination

See Figure 28-4.

1. Sensibility:
   - Pinprick (two-point discrimination): Normal is < 6 mm when the points are static and < 3 mm when the points are moving. Abnormal values seen with underlying nerve injury.
   - Immersion test: Skin on palm of hand should wrinkle within 10 minutes when immersed in water. Failure to do so suggests underlying nerve injury.

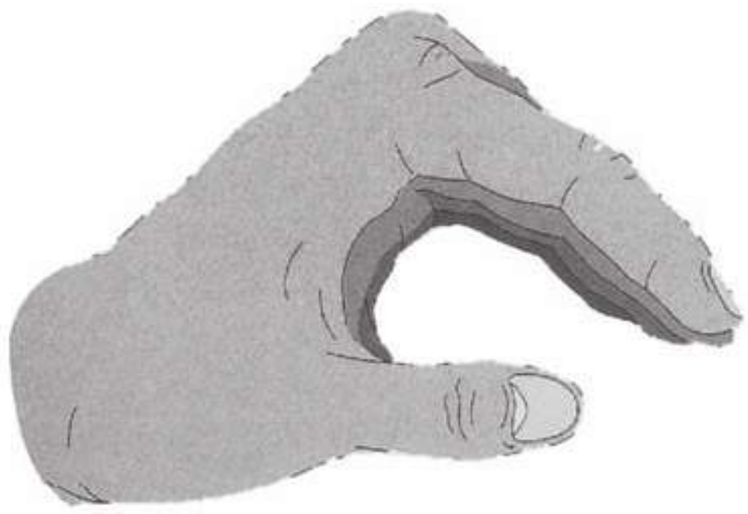


FIGURE 28-4. "Safe" position of hand.

2. Strength:
   - Test grip
   - Fromment's sign
3. Vascular:
   - Capillary refill: Normal is < 2 seconds.
   - Allen test:
     - Patient makes a tight fist for 20 seconds.
     - Examiner occludes both ulnar and radial arteries by holding direct pressure.
     - Examiner releases ulnar artery—a normal (patent) ulnar artery will perfuse the hand within 5–7 seconds (color returns).
     - Test is repeated with the radial artery released to check ulnar flow.
4. Motor and sensory function: See section under Nerves for which nerves supply which muscles and sensory areas.

WARD TIP

Flexor zone II (also called "no man's land") is the worst place for a tendon laceration because the tendon must slide in a small tunnel, and much care at surgery, as well as a long rehabilitation period, is required to achieve a good functional outcome.

Nerve Blocks

- Used to anesthetize a portion of the hand innervated by certain nerve(s) (see Figure 28-4).
- Advantages over local anesthesia:
  - Does not distort area you want to examine/suture.
  - Eliminates need for multiple injections.

Infections of the Hand

Felon

definition

Infection of the pulp space of any of the distal phalanges (see Figure 28-5).

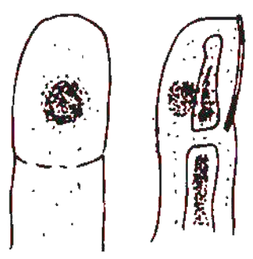


FIGURE 28-5. Felon (infection of pulp space). (Reproduced, with permission, from LeBlond RF, DeGowin RL, Brown DD. DeGowin's Diagnostic Examination. 8th ed. New York: McGraw-Hill; 2004: 729.)

etiology

Caused by minor trauma to the dermis over the finger pad.

Complications

Results in increased pressure within the septal compartments and may lead to cellulitis, flexor tendon sheath infection, or osteomyelitis if not effectively treated.

Treatment

- Using a digital block, perform incision and drainage (I&D) with longitudinal incision over the area of greatest induration but not over the flexor crease of the DIP.
- Drain may be placed and wound checked in 2 days.
- Antibiotics: Usually first-generation cephalosporin or anti-Staphylococcus penicillin.

Paronychia

definition

Infection of the lateral nail fold (see Figure 28-6).

etiology

Caused by minor trauma or activities such as nail biting or manicures.

EXAM TIP

Kanavel signs of tenosynovitis: **STeP**
Symmetrical swelling of finger.
Tenderness over flexor tendon sheath.
extension (passive) of digit is painful.
Posture of digit at rest is flexed.

Treatment

- Without fluctuance, this may be treated with a 7-day course of antibiotics, warm soaks, and retraction of the skin edges from the nail margin.
- For more extensive infections, unroll the skin at the base of the nail and at the lateral nail or I&D at area of most fluctuance using a digital block. Pus below the nail bed may require partial or total removal of the nail. Warm soaks and wound check in 2 days. Antibiotics are usually not necessary unless area is cellulitic.

sePTic Flexor TenosynoviTis

etiology

This is a surgical emergency requiring prompt identification. Infection of the flexor tendon and sheath is caused by penetrating trauma and dirty wounds (e.g., dog bite). Infection spreads along the tendon sheath, allowing involvement of other digits and even the entire hand, causing significant disability.

Organisms

- Polymicrobial.
- Staphylococcus most common.
- Neisseria gonorrhoeae with history of sexually transmitted disease (STD).



FIGURE 28-6. Paronychia. (Reproduced, with permission, from LeBlond RF, DeGowin RL, Brown DD. DeGowin's Diagnostic Examination. 8th ed. New York: McGraw-Hill; 2004: 729.)

Treatment

- Immobilize and elevate hand.
- Immediate consultation with hand surgeon.
- Parenteral antibiotics; first-generation cephalosporin and penicillin, or β-lactamase inhibitor.

Gamekeeper's Thumb

deFiniTion

Avulsion of ulnar collateral ligament of first MCP joint.

eTiology

- Forced abduction of the thumb.
- Can be associated with an avulsion fracture of the metacarpal base.

signs and syMPToMs

Inability to pinch.

diagnosis

Application of valgus stress to thumb while MCP joint is fexed will demonstrate laxity of ulnar collateral ligament.

Tr eaTMen T

- Rest, ice, elevation, analgesia.
- Thumb spica cast for 3–6 weeks for partial tears.
- Surgical repair for complete tears.

EXAM TIP

Gamekeeper's thumb is commonly associated with ski pole injury (see Figure 28-7).

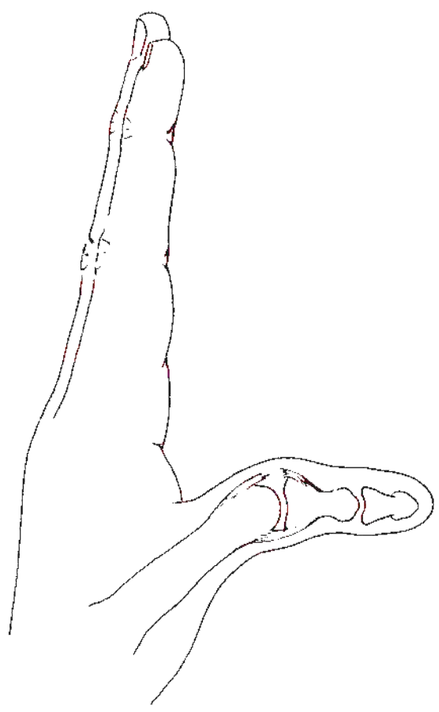


FIGURe 28-7. Gamekeeper's thumb. (Reproduced, with permission, from Scaletta TA et al. Emergent Management of Trauma. New York: McGraw-Hill; 1996: 220.)

EXAM TIP

Carpal tunnel syndrome is the most common entrapment neuropathy.

Carpal Tunnel Syndrome

Definition

Compression of the median nerve resulting in pain and sensory disturbances along the distribution of the nerve (see Figure 28-8).

Etiology

- Idiopathic/overuse (most common).
- Tumor (fibroma, lipoma).
- Ganglion cyst.
- Tenosynovitis of flexor tendons secondary to rheumatoid arthritis or trauma.
- Edema due to pregnancy, thyroid, or amyloid disease.
- Trauma to carpal bones.
- Gout.

Risk Factors

Repetitive hand movements.

EXAM TIP

Typical scenario: A 37-year-old female presents with pain in her right wrist and fingers, accompanied by a tingling sensation. The pain awakens her from sleep, and she is unable to perform her duties as a word processor. Think: Carpal tunnel syndrome.

Epidemiology

More common in women 3:1.

Signs and Symptoms

- Pain and paresthesia of volar aspect of thumb, digits 2 and 3, and half of digit 4.
- Activity and palmar flexion aggravate symptoms.
- Thenar atrophy: Uncommon but irreversible and indicates severe long-standing compression.
- Sensory deficit (two-point discrimination > 5 mm).

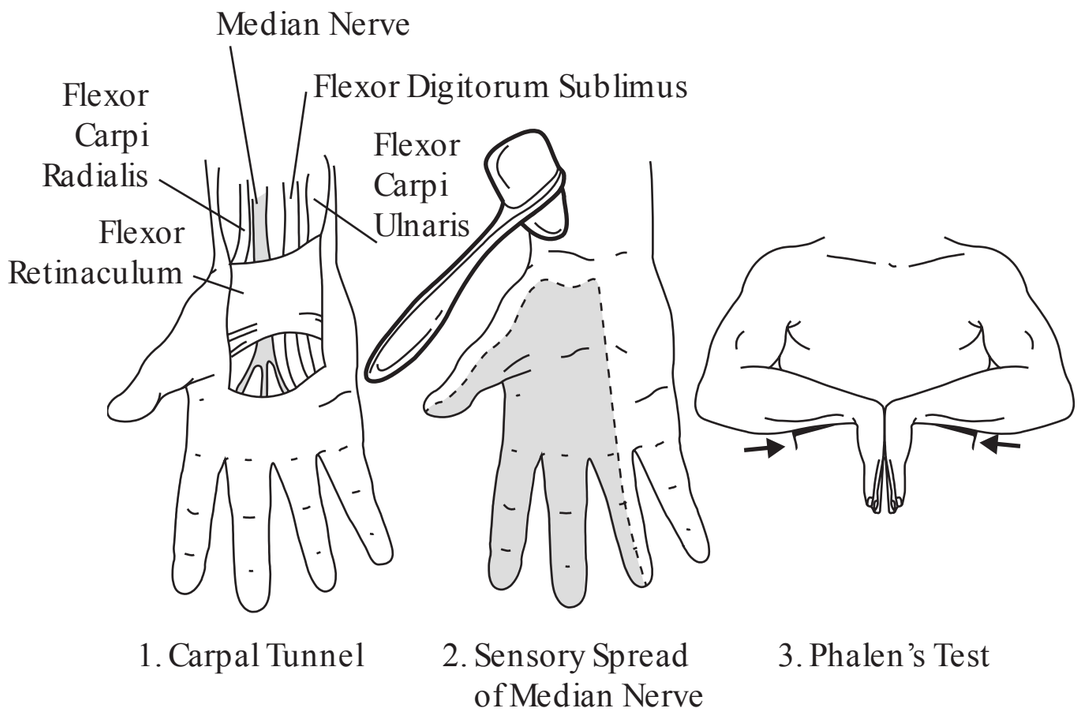


FIGURE 28-8. Carpal tunnel syndrome. (Reproduced, with permission, from LeBlond RF, DeGowin RL, Brown DD. DeGowin's Diagnostic Examination, 8th ed. New York: McGraw-Hill, 2004: 736.)

diagnosis

- Tinel's test: Tapping over median nerve at wrist reproduces pain and paresthesia.
- One minute of maximal palmar flexion reproduces pain and paresthesia.
- Consider erythrocyte sedimentation rate (ESR), thyroid function tests (TFTs), serum glucose, and uric acid level to look for underlying cause.

Treatment

- Treat underlying condition.
- Rest and splint.
- Nonsteroidal anti-inflammatory drugs (NSAIDs) for analgesia.
- Surgery for untreatable pain, thenar atrophy, and failure of nonoperative management.

EXAM TIP

Twenty to 50% of the population normally have a positive Phalen's and Tinel's test.

Ganglion Cyst

definition

A synovial cyst, usually present on radial aspect of wrist.

etiology

Idiopathic.

signs and symptoms

- Presence of mass that patient cannot account for.
- May or may not be painful.
- Pain aggravated by extreme flexion or extension.
- Size of ganglia increases with increased use of wrist.
- Compression of median or ulnar nerve may occur (not common).

diagnosis

Radiographs to ascertain diagnosis; since a ganglion cyst is a soft tissue problem only, no radiographic changes should be noted.

EXAM TIP

Differential diagnosis includes bone tumor, arthritis, and intraosseous ganglion.

Treatment

- Reassurance for most cases.
- Wrist immobilization for moderate pain.
- Aspiration of cyst for severe pain.
- Surgical excision for cases involving median nerve compression and cosmetically unacceptable ganglia.

Mallet Finger

definition

Rupture of extensor tendon at its insertion into base of distal phalanx (see Figure 28-9).

EXAM TIP

Left untreated, mallet finger results in permanent boutonniere deformity.



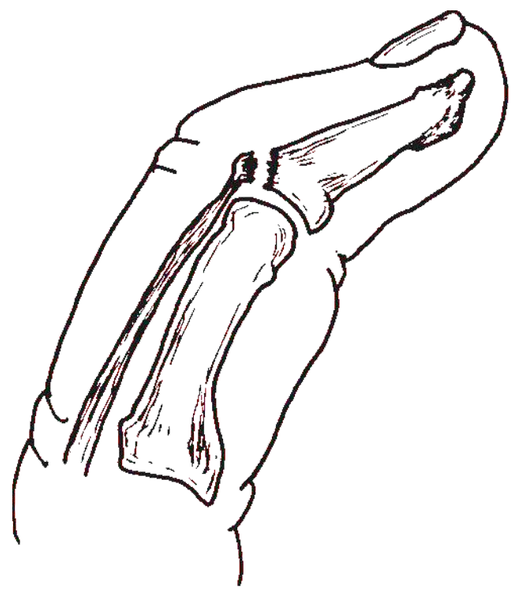


FIGURE 28-9. Mallet finger. (Reproduced, with permission, from Schwartz DT, Reisdorf EJ. Emergency Radiology. New York: McGraw-Hill; 2000: 40.)

ETIOLOGY

- Avulsion fracture of distal phalanx.
- Other trauma.

SIGNS AND SYMPTOMS

Inability to extend DIP joint.

TREATMENT

- Splint finger in extension for 6–8 weeks.
- Surgery may be required for large avulsions of distal phalanx and for injuries that were not splinted early.

Trigger Finger

DEFINITION

Stenosis of flexor digitorum tendon sheath (A1 pulley) leading to nodule formation within the sheath (see Figure 28-10).



FIGURE 28-10. Trigger finger. (Reproduced, with permission, from LeBlond RF, DeGowin RL, Brown DD. DeGowin's Diagnostic Examination. 8th ed. New York: McGraw-Hill; 2004: 727.)

Risk Factors

- Rheumatoid arthritis
- Middle-aged women
- Congenital

Signs and Symptoms

Severe pain and snapping sensation or click when flexing and extending the digit.

Treatment

- Splinting of MP joint in extension.
- Injection of corticosteroid into tendon sheath.
- Surgical release of the A1 pulley if above fail.

Amputation Injuries

Prognosis is best for:

- Sharp vs. crush or avulsion amputations.
- Children vs. adults.
- Clean vs. dirty.

Indications For Surgery

- Amputations with good prognosis as listed above.
- Thumb amputations (unlike other single digits, amputation of the thumb leaves a significant deficit in function of the hand).
- Multiple-digit amputations.

Relative Contraindications To Surgical Reconstruction

- Amputations in smokers (poor healing)
  - This is especially true if patient cannot commit to abstain from smoking for a 3-month period post reimplantation.
- Severe crush or avulsion injury.
- Grossly contaminated injuries.
- Amputations at multiple levels along the amputated limb.

Complications

- Stiffness.
- Cold intolerance.
- Decreased sensibility.
- Patients often require long-term hand therapy following surgery.

Preoperative Management

- IV antibiotics.
- Tetanus prophylaxis.
- NPO for possible surgery.
- Cleansing of wound site and amputated part with saline or lactated Ringer's.
- Wrap amputated part in sterile gauze.
- X-ray both limb and amputated part.

WARD TIP

Do not be fooled by how insignif cant a high-pressure injection injury to the hand appears on initial presentation.

High-Pressure Injection Injuries

- Commonly associated with grease guns, spray guns, and diesel fuel injectors.
- Usually occurs to index f nger of nondominant hand.

Sign S and Sympt omS

- Seemingly innocuous puncture wound at initial presentation.
- Edema and minimal pain progress to more severe pain, discoloration, and swelling, then to intense tissue necrosis within 24 hours.

diagnoSiS

Hand radiograph may reveal path of injected material if radiopaque.

t r eat ment

- IV antibiotics.
- Hospital admission for close observation.
- I&D—often operative; obtain hand surgery consult.
- Amputation may be necessary if part not salvageable.
- Physical therapy after acute management.

Other Common Hand and Wrist Injuries

See Table 28-4.

TABLE 28-4. Common Hand and Wrist Injuries

Injury	Description	treatment
Boxer's fracture	Fracture of neck of fifth metacarpal sustained in a closed fist injury	▪ Ulnar gutter splint with MCPs flexed to 90° and PIP fully extended for 3–6 weeks ▪ Surgical repair for: ▪ Any rotational deformity ▪ Angulation of fourth/fifth metacarpal > 40° ▪ Angulation of second/third metacarpal > 10–15°
Bennett fracture	Fracture-dislocation of base of thumb	▪ Initially immobilization in thumb spica cast ▪ Definitive treatment is with surgical fixation
Rolando fracture	Comminuted fracture of the base of the thumb	▪ Initially immobilization in thumb spica cast ▪ Definitive treatment is surgical fixation
Scaphoid fracture	▪ Most commonly caused by fall on outstretched hand ▪ Snuf box tenderness is classic ▪ May take up to 2 weeks to be seen on radiographs ▪ Often requires scaphoid view or MRI to see fracture	▪ Immobilization in thumb spica cast with wrist in neutral position for 12 weeks ▪ If no healing is seen by 4 weeks, likely surgical intervention since nonunion rate is very high due to tenuous blood supply

(continues)

TABLE 28-4. Common Hand and Wrist Injuries (continued)

Injury	Description	Treatment
Colles' fracture	▪ Distal radius fracture with dorsal angulation ▪ Most commonly caused by fall on outstretched hand ▪ "Dinner fork deformity" is classic	▪ Short arm cast for 4–6 weeks with volar flexion and ulnar deviation ▪ Surgical repair for: ▪ Open fracture ▪ Comminuted fracture ▪ Intra-articular displacement > 2 mm
Smith fracture	▪ Distal radius fracture with volar angulation ▪ Most commonly caused by direct trauma to dorsal forearm	Surgical repair needed for most cases
Galeazzi fracture	▪ Distal one third radial fracture with dislocation of distal radioulnar joint ▪ Commonly caused by fall on outstretched hand with forearm in forced pronation or direct blow to back of wrist	Surgical repair needed for most cases
Monteggia fracture	▪ Proximal one third ulnar fracture with dislocation of the radial head ▪ Commonly caused by fall on outstretched hand with forearm in forced pronation or direct blow to posterior ulna ▪ May note injury of radial nerve	▪ Surgical repair for adults ▪ Closed reduction for children (children can tolerate a greater degree of displacement)
Nightstick fracture	▪ Isolated fracture of the ulnar shaft	▪ Long arm cast for 3–6 weeks ▪ Surgical repair for: ▪ Angulation > 10° ▪ Displacement > 50%

Section iV

Classified: awards and OppOrtunities

AmericAn AssociAtion for the Surgery of trAumA 462

new york AcAdemy of medicine—ferdinAnd c. VAlentine Student reseArch grAnts in urology 462

oregon heAlth And Science uniVersity—cAmpAgnA ScholArship in neurologicAl Surgery 462

Society for clinicAl And VAsculAr Surgery—peter B. sAmuels essAy AwArd competition 462

Society for clinicAl And VAsculAr Surgery—AllAstAir kArmody poster competition 463

roswell pArk cAncer institute—Summer oncology reseArch progrAm 463

uniVersity of texAs m.d. Anderson cAncer center—medicAl Student Summer reseArch progrAm in BiomedicAl Sciences 463

sign AAn neurology medicAl Student progrAms 464

medicAl Student Summer reseArch ScholArship in neurology 464

AAn medicAl Student prize for excellence in neurology 465

medicAl Student essAy AwArd—rolAnd p. mAckAy AwArd 465

michAel s. pessin Stroke leAdership prize 466

medicAl Student essAy AwArd—extended neuroscience AwArd 466

medicAl Student essAy AwArd—g. milton Shy AwArd 467

medicAl Student essAy AwArd—SAul r. korey AwArd 468

uniVersity of cAliforniA At dAVis ophthAlmology Summer fellowship 468

hArVArd-longwood reseArch trAining in VAsculAr Surgery Summer reseArch fellowships in VAsculAr Surgery: 469

nyu hospitAl for Joint diseAses depArtment of orthopAedics (yeAr-long fellowship) 470

AmericAn otologicAl Society medicAl Student reseArch trAining fellowships 470

the nyu urology Summer fellowship 470

the WilliAm J. Von leiBig Summer reseArch fellowship in VAsculAr Surgery At hArVArd medicAl School 471

the AmericAn AssociAtion of neurologicAl Surgeons (AAns) through the neurosurgery reseArch And educAtion foundAtion (nref) 472

the orthopedic reseArch And educAtion foundAtion 472

uniVersity of WisconsiN–mAdison—Surgery Summer reseArch experience for medicAl Students 473

the cns/csns medicAl Student Summer fellowship in Socioeconomic reseArch 474

fight for Sight—Summer Student fellowships 474

the hospitAl for SpeciAl Surgery—medicAl Student Summer reseArch fellowship 475

the AmericAn AssociAtion of thorAcic Surgery—medicAl Student internship 475

American Association for the Surgery of Trauma

AAST provides medical student scholarships to attend the annual meeting. You may be sponsored by a member of the AAST to attend next year's annual meeting. You would be required to send a letter of recommendation by a member of the AAST for approval by the AAST Board of Managers. Once approved, your registration fee and banquet fee will be waived. The AAST will also cover the cost of your hotel room. In addition, you will be provided with an approximately $75 stipend for food/miscellaneous items. You/your institution are responsible for your travel arrangements. Contact the AAST for the deadline for medical student scholarship applications (CV and letter of recommendation from an AAST member), generally June of each year. For consideration, medical students should send their letters of recommendation and curriculum vitae (CV) to:

Robert C. Mackersie, MD
Trauma/Critical Care
UCSF-San Francisco General Hospital
1001 Potrero Avenue, Ward 3A
San Francisco, CA 94110
Phone: (415) 206-4622
Fax: (415) 206-5484
E-mail: rmackersie@sfghsurg.ucsf.edu

new york Academy of medicine—ferdinand c. Valentine student research grants in urology

Eligibility: Open to medical students who plan to conduct a New York-based research project in urology

Application Deadline/Start Date: Generally each February

Additional Information: www.nyam.org/

oregon health and science university—campagna scholarship in neurological surgery

Eligibility: Open to students who have completed their M1 or M2 year who are interested in participating in a mentored neurosurgical research project in Portland, Oregon.

Application Deadline/Start Date: Generally each February

Additional Information: www.ohsu.edu/ohsuedu/academic/som/neurosurgery/news-and-events/campagna-scholarship.cfm

society for clinical and Vascular surgery—peter B. samuels essay award competition

Open to students, residents, and fellows in a university, affiliated hospital, or in a surgical or vascular surgical residency in the United States or Canada. Submissions must be original papers on a subject related to vascular surgery that have not been previously published or presented. Abstracts may be based on either experimental or clinical observations or it may be analytical, based on analysis and review of previous published data on the anatomy, physiology, pathology, biochemistry, or genetics of the vascular system and its disease. Finalists for this award will be notified and must submit seven copies of the manuscript to the Society's office before the deadline specified for the annual

symposium. The 2009 award was $1,000 cash prize, plus round-trip coach airfare reimbursement and three nights' hotel accommodations.

society for clinical And VAscular surgery—Allast Air kArmody poster competition

First-round winners will each receive a cash award ($500 in 2009). The Karmody Poster Competition winner will receive an additional cash award.

- The competition is limited to fellows, residents, and medical students.
- All abstracts accepted for poster presentation will be considered. No additional f eld needs to be marked during the abstract submission process.
- Submissions must be original papers on a subject related to vascular surgery that have not been previously published or presented.
- Abstracts may be based on either experimental or clinical observations, or they may be analytical, based on analysis and review of previously published data on the anatomy, physiology, pathology, biochemistry, or genetics of the vascular system and its disease.

roswell pArk cAncer institute—summer oncology reseArch progrAm

Expand your horizons in the care and treatment of the cancer patient by participating in state-of-the-art clinical research. Explore the mysteries of the cancer cell by participating in a basic scientif c research program. Or participate in both! Special emphasis is placed on cancer prevention through lectures and practical experiences. The Roswell Park program provides competitive stipend support (projected at $350 per week) for students in the health professions (medicine, dentistry, and osteopathy) to engage in clinical and/or basic scientif c research for an 8-week period. Some funding is available to defray costs of room. The program runs from early June through the end of July (contact Roswell Park directly for specif cs); however, dates of participation may be changed to accommodate your academic schedule. **Applications:** Generally due in February of each year

Additional Information:
Arthur M. Michalek, PhD, FACE
Senior Vice President
Department of Educational Affairs
Roswell Park Cancer Institute
Carlton and Elm Streets
Buffalo, NY 14263
Phone: (716) 845-2339
E-mail: arthur.michalek@roswellpark.org

university of texAs m. d. Anderson cAncer center—medicAl student summer reseArch progrAm in BiomedicAl sciences

Research educational appointments for college students at the University of Texas M. D. Anderson Cancer Center provide f rsthand experience in the areas of cancer research and insight into the varied career opportunities available in the biomedical sciences. To qualify for a college student research appointment, the applicant must be classif ed as a college freshman, sophomore, junior, or nongraduate senior at the time of appointment. The applicant should be pursuing a career in sciences and have a transcript that demonstrates a record of academic achievement. The appointment policy limits an individual to a 1-year appointment, which may be renewed upon the

recommendation of the faculty mentor. College students seeking individual appointments should contact the faculty members or department administrators in the departments of interest to obtain information about available positions and the eligibility requirements of each program.

sign AAn neur ol ogy medicAl student pr ogr Ams

The Student Interest Group in Neurology (SIGN) program is a network of more than 150 chapters in medical schools across the United States and Canada. SIGN fosters medical student interest in neurology by providing opportunities to participate in clinical, research, and service activities in neurology, increasing the student's neurologic knowledge, and creating an interest in the AAN.

Free SIGN membership will enable you to:

- Socialize with students, residents, and faculty who share your interest in neurology
- Shadow neurologists
- Attend patient presentations and seminars
- Develop experience, leadership, and valuable contacts
- Join the nationwide SIGN network
- Meet other SIGN members at the Annual Meeting

Apply for SIGN scholarships:

- $3,000 Summer Research Scholarship
- $1,000 AAN Annual Meeting Scholarship

www.aan.com/go/education/awards

medicAl student summer r eseAr ch schol Ar ship in neur ol ogy

Application Deadline: Generally each February

Sponsored by the AAN's Undergraduate Education Subcommittee, the Medical Student Summer Research Scholarship program offers members of the AAN's Student Interest Group in Neurology (SIGN) program a summer stipend of approximately $3,000 to conduct a project in either an institutional, clinical or laboratory setting where there are ongoing programs of research, service or training, or a private practice. Only applicants from schools with established SIGN chapters are eligible to apply. The project is to be conducted through a U.S. or Canadian institution of the student's choice and jointly designed by the student and sponsoring institution. More than one student from an institution may apply, but only one student will be selected from an institution. The scholarship program was established to stimulate individuals to pursue careers in neurology in either research or practice settings.

The AAN will award up to twenty $3,000 scholarships to f rst or second-year AAN medical student members who have a supporting preceptor and a project with clearly def ned goals. Third-year AAN medical student members who are on an off cial summer break will also be considered with accompanying documentation. One graduating medical student from each institution is eligible to receive the award.

The project is to be conducted through a U.S. or Canadian institution of the student's choice and jointly designed by the student and sponsoring institution. Only applicants from schools with established SIGN chapters are eligible to apply. More than one student from an institution may apply, but only one student will be selected from an institution.

Applicants must be AAN medical student members at the time of application submission.

AAn medicAl student prize for excellence in neurology

Application Deadline: Generally each February

This award recognizes excellence in clinical neurology among graduating medical students. Only one medical student per institution should be nominated for the Medical Student Prize for Excellence. Awarded annually to a graduating medical student who exemplifes outstanding scientifc achievement and clinical acumen in neurology or neuroscience, and outstanding personal qualities of integrity, compassion, and leadership. A Certifcate of Recognition and a check for $200 will be presented on behalf of the AAN during the graduation or awards ceremony at each institution.

Application Procedure: Each department chair will designate a faculty committee that will select the award winner. The award winner will be selected based on outstanding performance in the neurology clerkship and outstanding personal and professional qualities, as noted in the award description.

Preference should be given to students choosing neurology as a career.

Submit material to calementi@aan.com, fax to (651) 361-4837 or mail to:

Medical Student Prize for Excellence in Neurology
Cheryl Alementi
American Academy of Neurology
1080 Montreal Avenue
St. Paul, MN 55116

Application Deadline: The deadline to nominate a medical student for the Medical Student Prize for Excellence is generally in February of each year.

medicAl student essAy AwArd—rolAnd p. mAckAy AwArd

This award seeks to stimulate interest in the feld of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in historical aspects.

Presentation: Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- Certifcate of Recognition and $350 prize
- Complimentary registration for 61st Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for 61st Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

Eligibility:
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists (essay cannot be a previously published manuscript for Mackay)
- Must have spent less than 1 year on a project leading to the submitted essay

Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):

- Must be typed, double-spaced using a standard font
- Maximum length of 30 pages
- Only deceased persons may be subjects of biographical papers
- Letter attesting to eligibility criteria and identifying award category
- Maximum 200-word abstract
- Letter from a faculty sponsor detailing the extent of technical or financial support received, the student's individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

michAel s. pessin stroke leAdership prize

Sponsored by the AAN and endowed by Dr. Pessin's family, friends, and colleagues, this award recognizes emerging neurologists who have a strong interest in and have demonstrated a passion for learning and expanding the field of stroke research. Applicants should have an active involvement in providing patients with the highest quality of compassionate care. This award is intended to stimulate and reward individuals in the developmental stages of their careers, who demonstrate a passion for stroke.

Presentation: Recipient is expected to give a 10-minute presentation on a topic of their choice during a scientific session at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $1,500 prize
- Complimentary registration for Annual Meeting
- Recognition at Awards Luncheon at Annual Meeting

Eligibility:
- Must be a medical student, resident, fellow, or junior faculty member involved in or considering a career in neurology, emphasizing the care of stroke patients
- Must be no more than 5 years from completion of most recent training program and no higher academic rank than assistant professor
- Additional consideration will be given to those involved in clinical research aimed at enhancing the understanding of stroke or improving acute treatment protocols

Application Procedure: Applicants should submit one complete set of the following materials:
- Completed application form
- Current curriculum vitae

For more information, please contact Franziska Schwarz at fschwarz@aan.com or (651) 695-2807.

medicAl student essAy AwArd—extended neuroscience AwArd

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in Neuroscience.

Presentation: Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- $1,000 prize
- Complimentary registration for Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

Eligibility:
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists
- Must have spent more than one year on a project leading to the submitted essay

Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
  - Must be typed, double-spaced using a standard font
  - Maximum length of 30 pages
  - Only deceased persons may be subjects of biographical papers
  - Letter attesting to eligibility criteria and identifying award category
  - Maximum 200-word abstract
  - Letter from a faculty sponsor detailing the extent of technical or financial support received, the student's individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

medical student essay award—g. milton shy award

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in clinical neurology.

Presentation: Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $350 prize
- Complimentary registration for Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

Eligibility:
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists (essay cannot be a previously published manuscript for Shy)
- Must have spent less than 1 year on a project leading to the submitted essay

Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
  - Must be typed, double-spaced using a standard font

- Maximum length of 30 pages
- Only deceased persons may be subjects of biographical papers
- Letter attesting to eligibility criteria and identifying award category
- Maximum 200-word abstract
- Letter from a faculty sponsor detailing the extent of technical or financial support received, the student's individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

medical student essay award—saul r. korey award

This award seeks to stimulate interest in the field of neurology as an exciting and challenging profession by offering highly competitive awards for the best essay. Essays are judged on the basis of the quality of the scholarship and on suitability for an audience of general neurologists.

This award is given for the best essay in experimental neurology.

Presentation: Recipients are expected to give a poster presentation based on the selected manuscript at the AAN Annual Meeting.

Recipient will receive:
- Certificate of Recognition and $350 prize
- Complimentary registration for Annual Meeting
- One-year complimentary subscription to Neurology journal
- Reimbursement for Annual Meeting travel, lodging, and meal expenses (up to 2 days)
- Recognition at Awards Luncheon at Annual Meeting

Eligibility:
- Must be enrolled and in good standing in an accredited medical school in North America
- Must submit an original, lucid essay targeted to general neurologists (essay cannot be a previously published manuscript for Korey)
- Must have spent less than 1 year on a project leading to the submitted essay

Application Procedure: Applicants should submit one complete set of the following materials:
- Completed application form
- Essay using the following guidelines (essays will not be returned):
  - Must be typed, double-spaced using a standard font
  - Maximum length of 30 pages
  - Only deceased persons may be subjects of biographical papers
  - Letter attesting to eligibility criteria and identifying award category
  - Maximum 200-word abstract
  - Letter from a faculty sponsor detailing the extent of technical or financial support received, the student's individual contribution to the project, and verifying that the student is the sole author of the essay

For more information, please contact Kyle Krause at kkrause@aan.com or (651) 695-2733.

university of california at davis ophthalmology summer fellowship

Department will offer summer fellowship stipend of $2,000 for expenses and supplies for a medical student to pursue a research project. Students first must find a project and a mentor for the summer project. Then the

student will need to write up the project proposal with a budget and submit it to the Department of Ophthalmology Research Committee. The committee will award the stipends based on the quality of the submitted proposal.

Additional funding besides a summer stipend for medical student research is available. Students first must find a project and a mentor for the project. Then the student will need to write up the project proposal with a budget and submit it to the Department of Ophthalmology Research Committee. The committee will award the stipends based on the quality of the submitted proposal.

The projects are of two types:

1. (Short Projects) These projects should be completed in a relatively short time frame (e.g., 2 to 3 months). A student could start it on a 4- to 8-week rotation and then write the project up at a later time. The type of projects that would be appropriate might be:
   a. Case reports
   b. Chart reviews on particular projects asking a limited and specific question
2. (Long Projects) These projects would be more long-range projects that would take 6 to 12 months. The student would be taking off from medical school to do the project.
   a. The projects may be prospective clinical projects with a well-defined question, mentor, research plan, and budget with funding defined.
   b. The project may be a laboratory project with a well-defined question, mentor, research plan, and budget with funding defined.

Web site: www.ucdmc.ucdavis.edu

Harvard-Longwood Research Training in Vascular Surgery summer research fellowships in vascular surgery:

- This experience is supported by the William J. von Liebig Summer Research Fellowship program.
- Four student research fellowships available starting (generally) on June 1.
- Program runs 10–12 weeks over the summer, with research training in molecular and cell biology, coagulation and thrombosis, atherogenesis, intimal hyperplasia, prosthetic/host interactions, and thrombosis.
- Trainees will pursue a program of intense research activity, which will be carried out under the guidance of a selected faculty advisor based at one of four Harvard Medical School hospitals.
- Applicants should have a minimum of 1 year of medical school at an LCME accredited school.
- Student will receive a $5,000 stipend for the summer.
- Application deadline is generally January.

For more information, contact:

Leena Pradhan, PhD
William J. von Liebig Summer Research Fellowship
Harvard Institutes of Medicine
4 Blackfan Circle, Room 130
Boston, MA 02115
Phone: (617) 667-0096
Fax: (617) 975-5300
E-mail: lpradhan@bidmc.harvard.edu

nyu hospital for joint diseases department of orthopaedics (year-long fellowship)

- One-year fellowship for medical students interested in pursuing a career in orthopaedics.
- The program is centrally located at the Hospital for Joint Diseases and Bellvue Medical Center with some travel to Jamaica Hospital in Queens, New York.
- Responsibilities will include participation in ongoing studies, maintenance of the Orthopaedic Trauma Service database, and submission of IRB protocols.
- Applicants should be in medical school or a recent graduate.
- A monthly stipend is given to help offset the cost of New York City housing, which is not provided.
- For more information, go to: www.med.nyu.edu/orthosurgery/research/opp.html

For more information, please contact:
Kenneth Egol
E-mail: kenneth-egol@nyumc.org

american otologic al society medical student research training fellowships

Purpose: To further the study otosclerosis, Ménière's disease, and related ear disorders.

The American Otologic Society, Inc., through its Research Fund, is offering Research Grant Awards and full time Research Training Fellowships to study otosclerosis, Ménière's disease, and related ear disorders in U.S. or Canadian institutions only. Proposals may include investigation of the management and pathogenesis of these disorders, and underlying processes.

Research Training Fellowship: For physicians only (residents and medical students), fellowship will support 1–2 years' full-time research conducted outside of residency training; $35,000 for stipend, $5,000 for supplies (and up to 10% indirect costs).

Applications must be accompanied by sponsoring institution documentation stating that facilities and faculty are appropriate for requested research. Research conducted during the Research Training Fellowship can be on any topic related to ear disorders.

Deadline: Grant and fellowship applications must be postmarked by January 31.

Information and materials may be obtained from www.americanotologicalsociety.org/forms.html or by contacting:

Lloyd B. Minor, MD, Executive Secretary
Research Fund of the American Otological Society, Inc.
Johns Hopkins University, School of Medicine
Department of Otolaryngology–Head & Neck Surgery
601 N. Caroline Street, JHOC 6210
Baltimore, MD 21287-0910
Phone: (410) 955-1080
Fax: (410) 955-6526
E-mail: lminor2@jhmi.edu

the nyu urology summer fellowship

This is an 8-week opportunity for two students between their first and second years of medical school to learn more about this exciting and diverse surgical subspecialty. This summer experience will allow students to spend time

in the operating room observing procedures in urologic oncology, female and male incontinence, infertility, erectile dysfunction, stone disease, and pediatric urology. Students will also spend time in the clinic observing urological outpatient procedures and office visits with patients before and after surgery. Summer fellows will attend teaching conferences and journal clubs run by faculty and residents.

To round out this exciting summer experience, students will be assigned to work with one of the Urology attending physicians on a clinical research project. For highly motivated students, this is a great opportunity to take a project from the start and create abstracts and manuscripts eligible for submission to national conferences and peer-reviewed journals for publication. Students will have the opportunity and are strongly encouraged to continue this research following completion of the summer fellowship. Finally, at the end of the fellowship, students will give a short oral presentation to the Department of Urology on a topic to be decided. This summer program will provide students with great exposure to this surgical subspecialty and research opportunities that can lead to medical conference presentations and publications.

Eligibility and Salary: Students must be between their first and second years of medical school to apply.

A stipend of $800 is provided. In addition to the stipend, students may receive additional funds in the amount of $2,100 through the NYU School of Medicine Office of Financial Aid if they qualify for work study. However, work-study eligibility is not required to apply.

How to Apply: Please forward a one-page cover letter addressed to Dr. William Huang, Director of the Urology Summer Fellowship, describing your interest in the fellowship, along with your resume, to Sabine Gay, program coordinator, at Sabine.Gay@nyumc.org. For further information, call (646) 825-6310.

Application Deadline and Important Dates: Students must submit applications by February 15 for the upcoming summer.

Notification of acceptance into this program will be in mid-March. The fellowship program runs from mid-June to early August.

the william J. von leibig summer research fellowship in vascular surgery at harvard medical school

Available June 1. Four medical student research fellowships are available for 10–12 weeks of summer research training in molecular and cell biology, biomechanics, coagulation and thrombosis, and angiogenesis, with a focus on clinically relevant problems such as atherogenesis, intimal hyperplasia, prosthetic/host interactions, and thrombosis. Trainees will pursue a program of intense research activity. This training program is designed to provide medical students an initial exposure to vascular surgery research.

Students will carry out their research projects under the guidance of a faculty advisor, selected form renowned vascular researchers based at four Harvard Medical School hospitals: Beth Israel Deaconess Medical Center, Brigham and Women's Hospital, Children's Hospital (Boston), and the Joslin Diabetes Institute, as well as the Massachusetts Institute of Technology.

Selection of trainees is based on candidates' demonstrated ability. Applicants should be medical students who have completed at least 1 year of study at an LCME accredited school. Students must be U.S. citizens or permanent residents (green card holders).

Interested applicants are encouraged to submit a personal statement, together with a curriculum vitae, dean/advisor or program director's letter, and two letters of recommendation. Selection is based on merit only, without bias to gender, race, color, or ethnic origin.

Support: A $5,000 stipend for the summer and appointment at Harvard Medical School as a Research Fellow in Surgery.

Student Program Director:
Frank W. LoGerfo, MD
Chief, Division of Vascular Surgery
Beth Israel Deaconess Medical Center
William V. McDermott Professor of Surgery
Harvard Medical School

Contact:
Leena Pradhan, PhD
William J. von Liebig Summer Research Fellowship
Harvard Institutes of Medicine
4 Blackfan Circle, Room 130
Boston, MA 02115
Tel: 617-667-0096
Fax: 617-975-5300
E-mail: lpradhan@bidmc.harvard.edu

the American Association of Neurological Surgeons (AANS) through the Neurosurgery Research And Education Foundation (NREF)

The new AANS Medical Student Summer Research Fellowship (MSSRF) program. The fellowship is open to medical students in the United States or Canada who have completed 1 or 2 years of medical school and wish to spend a summer working in a neurosurgical laboratory, mentored by a neurosurgical investigator who is a member of the AANS and will sponsor the student.

This year, 15 Medical Student Summer Research Fellowships will be awarded in the amount of $2,500 per award.

To be considered for this award, applications need to be received by February 1. Awardees will be notified and posted on the AANS web site by March 31.

Submit completed applications to:

AANS Medical Student Summer Research Fellowship
c/o American Association of Neurological Surgeons
5550 Meadowbrook Drive
Rolling Meadows, IL 60008-3852

or e-mail application and all supporting documents to nref@aans.org.

For more information about this medical student summer research fellowship program, contact the Development Department, toll free at (888) 566-2267 or info@aans.org.

the Orthopedic Research And Education Foundation

The Orthopaedic Research and Education Foundation sponsors a Medical Student Summer Orthopaedic Research Fellowship. Our goal is to encourage medical students considering a career in orthopaedics to gain experience in basic, clinical, or translational research.

The Orthopaedic Research and Education Foundation (OREF) is an independent organization that raises funds to support research and education on diseases and injuries of bones, joints, nerves, and muscles. OREF-funded research enhances clinical care, leading to improved health, increased activity, and a better quality of life for patients.

Fellowship Description:

- Medical students with an interest in orthopaedics are eligible to apply.
- The medical student needs to identify an investigator at a U.S. institution with an ongoing orthopaedic research project who is willing to provide research training to the student and act as his/her mentor.
- OREF will provide $2,500 as salary support for the student, payable directly to the sponsoring institution. OREF will reimburse the institution for FICA taxes of up to $200 and up to $200 for supplies, if requested. No other fringe benefits are authorized.
- This program is intended to be a summer research fellowship. A minimum of 8 weeks' full-time work on a specific project is required. The research project should be one that the student is not already involved in.
- At the completion of the program, the medical student is required to complete an evaluation form provided by OREF. The mentor will also complete an evaluation form.

Orthopaedic Research and Education Foundation (OREF)
6300 North River Road, Suite 700
Rosemont, IL 60018-4261
847-698-9980/847-698-9981
Web site: http://www.oref.org

University of Wisconsin–Madison—Surgery Summer Research Experience for Medical Students

The decrease in the number of physicians interested in pursuing a career as a clinician-scientist is well documented in lower numbers of NIH applications and in AAMC questionnaires. The Department of Surgery at the University of Wisconsin is uniquely qualified to direct a short-term summer research experience for medical students who are interested in research related to diabetes, obesity, endocrine disorders, nutritional disorders, digestive diseases, liver disease, kidney disease, and urologic disease. The overall program goal of this Department of Surgery training program is to provide six medical students with a focused, mentored 12-week research and training experience that will help students discern their career path, preferably toward a career that involves biomedical research. During this experience, all students will plan and complete a research project, complete a learning contract, attend an established curriculum in effective research and academic conduct, and write an abstract for submission and presentation at a medical meeting. The training program will foster the development of knowledge, competence, skills, professional attitudes, and experience required to understand what is involved in successful academic careers in laboratory or clinically based research related to the goals of the NIDDK. The four specific objectives of this program are to:

1. Expose medical students, early in their training, to the excitement and challenges of a research career through participation in an individual, mentored training experience.
2. Encourage students to pursue a research career through participation in a focused 12-week research experience in a research program of relevance to the mission of NIDDK.
3. Provide a didactic curriculum emphasizing research issues.

4. Increase the pool of medical students who pursue further research activities by partnering medical students with physicians and scientists who will serve as role models for a career combining patient care and scientific research. The training program includes plans to follow up with trainees later in their career to evaluate whether their summer surgical research experience in the Department of Surgery led to additional research training and a career as a clinician-scientist.

Contact: Herbert Chen, MD, University of Wisconsin–Madison

the cns/csns medical student summer fellowship in socioeconomic research

Award Amount: $2,500

Number of Awards: 2

Purpose: The CNS/CSNS Medical Student Socioeconomic Fellowship supports a medical student conducting research on a socioeconomic issue impacting neurosurgical practice.

Eligibility: The fellowship is open to all medical students in the United States and Canada. The fellow will spend 8–10 weeks conducting supervised research on a socioeconomic topic of importance to neurosurgery.

Requirements: The fellow must submit a final report to the CNS Fellowships Committee by December 1 following completion of the summer fellowship. Any publications resulting from supported research must acknowledge the support.

Eligible Expenses: Financial support is exclusively for stipend support to the student.

Application Requirements:
- Complete the online CNS/CSNS Medical Student Fellowship Application.
- Two reference letters.
- Curriculum vitae of applicant.
- Curriculum vitae of proposed mentor.
- Attach a headshot photograph.

Congress of Neurological Surgeons
Elad I. Levy, MD
Chairman, CNS Fellowships Committee
10 North Martingale Road, Suite 190
Schaumburg, IL 60173
Phone: (847) 240-2500
Fax: (847) 240-0804
Toll Free: (877) 517-1CNS
E-mail: info@1CNS.org

fight for sight—summer student fellowships

Awards of $2,100 are offered to currently enrolled undergraduates, medical students, or graduate students who wish to explore ophthalmology or eye research as a career. Students are expected to complete a short, independent project during the summer months under the guidance of a senior scientist or clinician. The goal of this award is to advance the skills needed to initiate and carry out research in a scientific environment. Lab employees are not eligible for this fellowship.

Go to www.fightforsight.com/grant_IGP.php for more information.

the hospital for special surgery—medical student summer research fellowship

This is an 8-week program of mentored research designed to introduce students who have completed their first year of medical school to research opportunities in orthopaedic basic science, translational science, and clinical research in orthopaedics.

The fellowship program is built around a summer research project that is directed by an orthopaedic surgeon or scientist at the Hospital for Special Surgery (HSS). Both clinical and basic science projects are offered, and these are detailed below.

The fellowship program includes the opportunity for weekly observation of orthopaedic surgical procedures with a variety of clinical faculty. In addition, a weekly seminar series and discussion group provides the opportunity for students to expand their understanding of the fundamentals of medical research and clinical orthopaedics.

The 8-week program runs from late June through mid-August (in 2009: June 22 to August 14) and includes a $2,400 stipend. Up to 15 fellowship awards will be granted.

To apply for the program, eligible students should first contact an orthopaedic surgeon or scientist who is offering a research project that is of interest (see list below). Once the student has been accepted by the surgeon or scientist, the student and mentor may apply for the fellowship together. Alternatively, if a student has an established working relationship with a faculty member, projects not listed can be submitted for consideration.

Students who have completed their first year in an accredited U.S. medical school are eligible to apply. Preference will be given to those students attending Weill Cornell Medical College.

Download the Fellowship Application Form.

The applications must be sent to Lizandra Portalatin (portalatinl@hss.edu) by April 3. Students and mentors will be notified of the award by May 1.

For questions, contact Chisa Hidaka, MD, Assistant Scientist, Hospital for Special Surgery, at hidakac@hss.edu or (212) 774-2384.

Fellowship Coordinators: Chisa Hidaka, MD, and Jo A. Hannafin, MD, PhD

the american association of thoracic surgery—medical student internship

The Summer Intern Scholarship Program was established in 2007 to introduce the field of cardiothoracic surgery to first- and second-year medical students from North American medical schools. In the 2 years since its inception, the Summer Intern Scholarship has offered 100 medical students the opportunity to broaden their educational experience by providing scholarships to spend 8 weeks during the summer working in an AATS member's cardiothoracic surgery department.

Web site: www.aats.org/MSSR/medicalStudents.html

Index

5-fluorouracil (5-FU), 160
5Ps of compartment syndrome, 290
5Ws of postoperative fever, 31
6Ps of acute arterial insuff ciency, 304
6Ps of arterial insuff ciency, 59
8Ps of rapid-sequence intubation, 296

A

AAA. See Abdominal aortic aneurysm (AAA)
AAN prize for excellence in neurology, 465
AANS medical student summer research fellowship (MSSRF), 472
AAST liver injury scale. See American Association for Surgery of Trauma (AAST) liver injury scale
AAST spleen injury scale. See American Association for Surgery of Trauma (AAST) spleen injury scale
ABCs of shock, 62
ABCs of trauma care, 44–45
Abdominal aortic aneurysm (AAA), 39, 312–314
Abdominal CT anatomy, 40f
Abdominal pain. See Acute abdomen
Abdominal trauma, 50–56
  angiography, 54
  bowel injury, 55–56
  CT scanning, 54
  diagnostic peritoneal lavage (DPL), 53–54, 56
  exploratory laparotomy, 54
  FAST, 51–52, 52f, 53f
  laparoscopy, 54
  liver injury. See Hepatobiliary system
  physical examination, 50–51
  rule of 1/3s for abdominal stab, 50
  serial hematocrits, 54
  splenic trauma. See Spleen
Abdominal wall defects, 324–326
Abdominal wall problems. See Hernia and abdominal wall problems
ABI. See Ankle-brachial index (ABI)
Above-knee amputation (AKA), 309
Acalculous cholecystitis, 205–206
Acetazolamide, 369
Achalasia, 93–94, 95t
Acid-based disorders, 89–90, 89t
Acid burns, 98, 290
Acinar cells, 217
Acinic cell carcinoma, 354
Acoustic apparatus, 336–337
Acoustic neuroma, 339–340
Acquired umbilical hernia, 182–183
Acromegaly, 255, 372
ACS. See Acute compartment syndrome (ACS)
ACTH. See Adrenocorticotropic hormone (ACTH)
Acute abdomen, 35–41
  abdominal CT anatomy, 40f
  bleeding, 38t
  def ned, 36
  diagnosis, 37–38
  history, 36–37
  inflamed mass/abscess, 38t
  ischemic or strangulated bowel, 38t
  left lower quadrant (LLQ) pain, 39
  left upper quadrant (LUQ) pain, 39
  nonsurgical causes of abdominal pain, 41
  paralytic ileus, 38t
  perforated viscus, 38t
  peritonitis, 38t
  physical exam, 37, 37t, 38t
  right lower quadrant (RLQ) pain, 39
  right upper quadrant (RUQ) pain, 38–39
  surgical causes of abdominal pain, 38–41
  urgent operation, indications for, 41t
Acute arterial insuff ciency, 304
Acute arterial occlusion, 304–306
Acute bacterial rhinosinusitis, 342
Acute cholangitis, 207
Acute compartment syndrome (ACS), 316–317
Acute mesenteric ischemia (AMI), 139–140
Acute myeloid leukemia (AML), 260t
Acute myocardial infarction (AMI), 384
Acute otitis media (AOM), 338
Acute pancreatitis, 218–220
Acute respiratory distress syndrome (ARDS), 71–72
Acute scrotum, 417–418
ACV. See Assist-control ventilation (ACV)
Addison's disease, 250
Adenocarcinoma
  appendix, 174
  esophageal carcinoma, 102
  pancreas, 222–223
  salivary glands, 354
  small intestine, 136t
  stomach, 117–120
Adenoid cystic carcinoma, 354
Adenoma
  adrenal gland, 247
  pituitary gland, 254–255
  small intestine, 135t
Adenomatous polyps, 121
ADH. See Antidiuretic hormone (ADH)
Adjustable gastric banding, 117f
Adrenal androgens, 233t
Adrenal cortical carcinoma, 247–248
Adrenal gland, 246–252
  Addison's disease, 250
  adenoma, 247
  adrenal cortical carcinoma, 247–248
  anatomy, 246
  Cushing's syndrome, 248–249, 249t
  hyperaldosteronism, 251
  hyperplasia, 247
  hypoaldosteronism, 252
  pathways of adrenal steroid synthesis, 247f
  physiology, 246
Adrenal insuff ciency, 250
Adrenocorticotropic hormone (ACTH), 230t
Adrenoreceptor agonists, 68t
Advanced trauma life support (ATLS), 44–45
AEIOU, 87
Afferent loop syndrome, 114
Afrin, 342
Agnogenic myeloid metaplasia, 260t
Air conduction/bone conduction, 337
AKA. See Above-knee amputation (AKA)
Alanine transaminase (ALT), 187
Alcoholic liver disease, 409
Aldosterone, 233t
Alkali burns, 98
Alkaline phosphatase, 187
Alkaline reflux gastritis, 114
Allastair Karmody poster competition, 463
Allograft, 402
Alpha agonists, 68t
ALT. See Alanine transaminase (ALT)
Amebic abscess, 192
American Association for Surgery of Trauma (AAST) liver injury scale, 190t
American Association for Surgery of Trauma (AAST) spleen injury scale, 262t
American Association for the Surgery of Trauma scholarship, 462
American Association of Thoracic Surgery medical student internship, 475
American Otological Society research fellowship, 470
AMI. See Acute mesenteric ischemia (AMI); Acute myocardial infarction (AMI)
Amide anesthetics, 300
AML. See Acute myeloid leukemia (AML)
AMPLE history, 46
Ampulla of Vater, 125, 216
Amputation, 309–310, 457

Anal canal, 144f. See also Colon, rectum, and anal canal
Anal cancer, 165–166
Anal fissure, 163
Anaphylactic shock, 63–65
Anaphylaxis, 65
Anaplastic thyroid cancer, 242–243t
Anastomotic aneurysm, 311
"Anchovy paste" appearance, 192
ANDI classification of benign breast disorders, 271t
Androgens, 233t
Anesthesia, 295–300
  anatomy, 298
  ASA classification of surgical patients, 296t
  complications, 299t
  definition, 296
  emergence, 298
  epidural, 298, 299t
  induction anesthetics, 296, 297t
  local, 299
  maintenance, 298
  malignant hyperthermia, 299–300
  neuromuscular blockade, 296–297
  pediatric airway, 298, 298t
  spinal, 298, 299, 299t
  stages, 296
Anesthetic agents, 297t
Aneurysm, 311–314
Angina pectoris, 384
Angiogenesis, 26
Angiography, 54
Angiotensin I, 251
Angiotensin II, 251
Anion gap, 90
Anion gap metabolic acidosis, 88, 89
Ankle-brachial index (ABI), 303
Annular pancreas, 216
Anorectal abscess, 163
Anorectal angle, 145
Anorectal fistula, 164
Antecubital fossae, 45
Anterior cord syndromes, 376
Anterior rhinoscopy, 342
Anterior shoulder dislocation, 433, 433t, 434
Antidiuretic hormone (ADH), 231t
Antithymocyte globulin (ATG), 404
Anus, 145
Anus injury, 56
AOM. See Acute otitis media (AOM)
Aorta, crossclamping, 52
Aortic arch aneurysm, 395, 398
Aortic balloon valvuloplasty, 389
Aortic regurgitation, 389–391
Aortic stenosis, 18, 388–389
Aortoiliac disease, 306–307
Appendectomy, 171f, 172f
Appendiceal abscess, 174
Appendiceal neoplasms, 173–174
Appendicitis, 38, 169–172. See also Appendix
Appendicolith, 171f
Appendicular artery, 168
Appendix, 144f, 167–174
  acute appendicitis, 169–172
  adenocarcinoma, 174
  anatomy/structure, 168, 168f
  appendiceal abscess, 174
  appendiceal neoplasms, 173–174
  carcinoid syndrome, 173
  elderly patients, 173
  embryology and development, 168
  immunocompromised patients, 173
  mucinous tumors, 174
  NOTES, 174
  pregnant patients, 173
Apple core lesions, 160f
ARDS. See Acute respiratory distress syndrome (ARDS)
Arterial occlusion, 304
Arterial wall, 302
Arteriography, 303–304
ASA classification of surgical patients, 296t
Ascending aorta aneurysm, 395
Ascending arch aneurysm, 398
Ascending cholangitis, 206, 207
Ascending colectomy, 162f
Ascending colon, 144f
Ascites, 199
Aspartate transaminase (AST), 187
Aspirin, 24
Assessing the patient. See Surgical patient
Assist-control ventilation (ACV), 71t
AST. See Aspartate transaminase (AST)
Astrocytoma, 371
Atelectasis, 20, 31
ATG. See Antithymocyte globulin (ATG)
Atheroembolism, 305
Atherosclerotic aneurysm, 398
Atlantoaxial subluxation, 375f
ATLS. See Advanced trauma life support (ATLS)
Auerbach plexus, 125
Auricle, 336, 336f
Auscultatory triad of mitral stenosis, 386
Autograft, 402
AVPU, 44
Awards and opportunities, 461–475. See also Awards and opportunities (subject matter)
  AAN prize for excellence in neurology, 465
  AANS medical student summer research fellowship (MSSRF), 472
  American Association for the Surgery of Trauma scholarship, 462
  American Association of Thoracic Surgery medical student internship, 475
  American Otological Society research fellowship, 470
  Campagna scholarship in neurological surgery, 462
  CNS/CSNS summer fellowship in socioeconomic research, 474
  Fight for Sight summer student fellowship, 474
  Havard-Longwood summer research fellowship in vascular surgery, 469
  Hospital for Special Surgery summer research fellowship, 475
  Karmody poster competition, 463
  medical student essay award (extended neuroscience award), 466–467
  medical student essay award (Korey award), 468
  medical student essay award (Mackay award), 465–466
  medical student essay award (Shy award), 467–468
  medical student summer research scholarship in neurology, 464
  NYU Hospital for Joint Diseases Department of Orthopaedics year-long fellowship, 470
  NYU urology summer fellowship, 470–471
  Orthopedic Research and Education Foundation fellowship, 472–473
  Pessin stroke leadership prize, 466
  Rosewell Park Cancer Institute summer oncology research program, 463
  Samuels essay award competition, 462–463
  SIGN AAN neurology program, 464
  University of California at Davis ophthalmology summer fellowship, 468–469
  University of Texas M.D. Anderson Cancer Center summer research program in biomedical sciences, 463–464
  University of Wisconsin–Madison surgery summer research experience, 473–474
  Valentine student research grant in urology, 462
  von Leibig summer research fellowship in vascular surgery, 471–472
Awards and opportunities (subject matter), 461–475
  biomedical research, 473–474
  biomedical sciences, 463–464
  essay competition, 462–463, 465–468
  neurological surgery, 462
  neurology, 464, 465, 472
  neuroscience, 466–467
  oncology, 463
  ophthalmology, 468–469, 474
  orthopedics, 470, 472–473, 475
  otosclerosis, 470
  poster competition, 463

socioeconomic research, 474
stroke research, 466
thoracic surgery, 475
trauma, 462
urology, 462, 470–471
vascular surgery (essay competition), 462–463
vascular surgery (poster competition), 463
vascular surgery (summer research fellowship), 469
vascular surgery (von Leibig fellowship), 471–472
Axillary dissection, 267f, 268
Axillary tail of Spence, 266
Azathioprine, 404

B

Back pain, 437–438, 439
Balloon tamponade, 97
Balloon valvuloplasty, 387, 389
Band ligation, 97
Bare area, 187
Bariatric surgery, 116, 117f
Barium esophagram, 103
Barium swallow, 93
Barrett's esophagus, 102
Basal energy expenditure (BEE), 88
Bassini's repair, 180
Battle's sign, 365
"Beads on a string" appearance, 208
Beck's tamponade triad, 48
Beckwith-Wiedemann syndrome, 333
BEE. See Basal energy expenditure (BEE)
Beger procedure, 222
Bell-clap deformity, 414
Bell's palsy, 340
Below-knee amputation (BKA), 309, 310
Benign prostatic hyperplasia, 422–423
Bennett fracture, 458t
Bernoulli's principle, 311
Beta agonists, 68t
Bile, 188–189
Biliary atresia, 327–328
Biliary colic, 201
Biliary obstruction, 222
Biliary system, 200, 201f. See also Hepatobiliary system
Biliary tree anatomy, 200, 201f
Biliopancreatic diversion, 116, 117f
Bilious vomiting, 328
Bilirubin, 189t
BIRADS. See Breast imaging and reporting and data system (BIRADS)
Bismuth classification of hilar cholangiocarcinoma, 212f
BKA. See Below-knee amputation (BKA)
Bladder, 415–416
Bladder irrigation, 409
Bladder rupture, 58
Blue toe syndrome, 305
Blumer's shelf, 118
Blunt cardiac trauma, 48
BMI. See Body mass index (BMI)
Body fluids, 74, 75f
Body mass index (BMI), 21, 22, 116
Boerhaave syndrome, 97, 98, 115, 116
Bone tumors, 438–444
Botox, 94
Bowel anatomy, 144f
Bowel injury, 55–56
Bowel preparation, 24
Bowen's disease, 165
Boxer's fracture, 458t
Brain death, 402
Brain tumors, 370–374
Branchial apparatus anomalies, 357–358
Branchial arches, 357t
Branchial cleft cyst, 320–321
BRCA1/BRCA2, 276
Breast, 265–282
anatomy, 266, 266f
ANDI classification of benign breast disorders, 271t
axillary dissection, 267f, 268
benign disease, 271–273
blood supply, 266–267
cancer, 275–282. See also Breast cancer
cystosarcoma phyllodes, 273
differential diagnosis, 269–270
ductal carcinoma in situ (DCIS), 275t
fat necrosis, 270–271
fibroadenoma, 271–272
fibrocystic changes, 272
gynecomastia, 273, 274
hidradenitis suppurativa, 270
infectious/inflammatory conditions, 270–271
infiltrating ductal carcinoma, 274
infiltrating lobular carcinoma, 274
inflammatory carcinoma, 274
initial evaluation, 268, 269f
intraductal papilloma, 273
lobular carcinoma in situ (LCIS), 275t
lymphatic drainage, 267
malignant tumors, 274
mammary duct ectasia, 272
mastectomy, 268, 278
mastitis, 270
Mondor's disease, 272
nerves, 268t
nipple discharge, 273–274
Paget's disease, 274
palpable breast mass, 269–270, 269t, 270t
peau d'orange, 268
phyllodes tumor, 273
plasma cell mastitis, 272
premalignant disease, 275t
quadrants, 266
sclerosing adenosis, 273
self-examination, 268
Breast cancer, 275–282
BRCA1/BRCA2, 276
breast reconstruction, 281
chemotherapy, 281
diagnosis, 277–278
epidemiology, 275
genetic predisposition, 276
hormonal therapy, 280–281
hormone receptor status response to therapy, 280t
males, 282
mammography, 277, 277f
metastasis, 282
pregnant and lactating women, 282
prognosis, 282
recurrence, 281
risk factors, 276
screening recommendations, 276, 276t
staging system, 280t
TNM staging, 279t
treatment decisions, 278, 279t, 280t, 281f
types of operations, 278–280
Breast imaging and reporting and data system (BIRADS), 277
Breast reconstruction, 281
Breast self-examination, 268
Brodie's abscess, 437
Bronchoalveolar cancer, 392
Bronchoplasty, 394
Brown-Séquard syndrome, 376–377
Buck's fascia, 414
Bullet presentation, 5
BUN, 21
Burn scar cancer, 291
Burns, 283–291
antimicrobial agents, 286t
carbon monoxide poisoning, 289
chemical injury, 290–291
daily burn care, 286
debridement and skin grafting, 288
definitive therapy, 287
electrical injury, 289–290
extent/severity, 284–285, 285f
fluid resuscitation, 285–286
Galveston formula, 285
infection, 286, 288–289
inhalational injury, 289
Marjolin's ulcer, 291
nutrition, 291
operations, 287–288
outpatient treatment, 285
Parkland formula, 285
referral to burn center, 284
rule of nines, 284, 285f
treatment outline, 284
Bursitis, 437

C

C di*ff* colitis, 22, 148
CA 19-9, 223
CABG. See Coronary artery bypass grafting (CABG)
Calcitonin, 231t
Calcium balance, 83t, 85–87
Calcium sign, 396
Calories requirements, 88
Calot's triangle, 200
Campagna scholarship in neurological surgery, 462
Cantlie's line, 187
Caput medusa, 200
Carbon monoxide, 289
Carbon monoxide poisoning, 289
Carboxyhemoglobin, 290
Carcinoembryonic antigen (CEA), 223
Carcinoid syndrome, 137–138, 173
Carcinoma ex pleomorphic, 354
Carcinoma of esophagus, 102–105
Carcinoma of gallbladder, 209–210
Cardiac graft vasculopathy, 411
Cardiac output (CO), 62
Cardiac transplant, 410–411, 412
Cardiogenic shock, 65–66, 66t
Cardiothoracic surgery, 381–389
  aortic regurgitation, 389–391
  aortic stenosis, 388–389
  coronary artery bypass grafting (CABG), 383–385
  ischemic heart disease, 382–383
  lung cancer, 391–395
  mitral insuff ciency, 387–388
  mitral stenosis, 385–387
  NYHA classif cation, 383
  operative risk, 385
  paraneoplastic syndromes, 393
  percutaneous transluminal coronary angioplasty (PTCA), 384–385
  thoracic aortic aneurysm, 395–398
  thoracoabdominal aneurysm, 398–399
  tricuspid stenosis, 391
  valve prostheses, 391
  valvular heart disease, 385–391
  wide pulse pressure, 390
Career development. See Surgery clerkship
Carotid angiogram, 308
Carotid artery stenosis, 308–309
Carotid body tumor, 373
Carotid bruit, 308
Carotid endarterectomy (CEA), 308, 309
Carpal bones, 447
Carpal tunnel syndrome, 380, 454–455, 454f
Carpentier's classif cation of mitral insuff ciency, 387
Caustic injury, 98–99
Cavernous hemangioma, 193
CBD. See Common bile duct (CBD)
CBD obstruction. See Common bile duct (CBD) obstruction
CBD stones, 206–207. See Common bile duct (CBD) stones
CBW. See Current body water (CBW)
CCAM. See Congenital cystic adenomatoid malformation (CCAM)
CCK. See Cholecystokinin (CCK)
CDH. See Congenital diaphragmatic hernia (CDH)
CEA. See Carcinoembryonic antigen (CEA); Carotid endarterectomy (CEA)
Cecal volvulus, 155
Cecum, 144f
Celiac plexus block, 216
Central cord syndrome, 377
Central line placement, 316
Central nervous system (CNS) tumors, 370–371
Central nervous system (CNS) vasculature, 362
Central splenorenal shunt, 198f
Centroacinar and ductal cells, 217
Cerebral concussion, 365
Cerebral contusion, 366
Cerebral perfusion pressure (CPP), 364
Cerebrospinal fluid (CSF), 364
Cervical pharyngocricoesophageal myotomy, 96
Cervical spine f lms, 377
Cervical spine fracture, 377–379
Chance fracture, 50, 380
Charcot's triad, 206
Chemical injury, 290–291
Chest radiograph (CXR), 5–7
Child-Pugh score, 196t
Children. See Pediatric surgery
CHL. See Conductive hearing loss (CHL)
Cholangiocarcinoma, 211–213
Cholangiogram, 187
Cholangitis, 207
Cholecystectomy, 202, 206
Cholecystitis, 202–206
Cholecystokinin (CCK), 127t
Choledochal cyst, 210–211, 211f
Choledocholithiasis, 206–207
Cholelithiasis, 201–202
Cholesteatoma, 339
Cholestyramine, 148
Chondrosarcoma, 442
Chronic ischemia, 306–309
Chronic lymphocytic leukemia, 260t
Chronic mesenteric ischemia (CMI), 140–141
Chronic myeloid leukemia, 260t
Chronic myelomonocytic leukemia, 260t
Chronic pancreatitis, 220–222
Chronic sinusitis, 342
Chronic stable angina, 384
Cimetidine, 114
Circle of Willis, 363, 363f
Circumferential radial margin (CRM), 160
Cirrhosis, 197
Clay shoveler's fracture, 379, 380f
Clean-contaminated wound, 27
Clean wound, 27
Clerkship. See Surgery clerkship
Clerkship grade, 8–9
Clinical clerkship examination, 11–12
Clopidogrel, 24
Clostridium di*ff* cile colitis, 22, 148
CMI. See Chronic mesenteric ischemia (CMI)
CMV ulcer. See Cytomegalovirus (CMV) ulcer
CNS/CSNS summer fellowship in socioeconomic research, 474
CNS tumors. See Central nervous system (CNS) tumors
CNS vasculature. See Central nervous system (CNS) vasculature
Cockcroft Gault equation, 20
Coffee grounds, 115
Colitis, 148–151
Colles' fracture, 459t
Colloids, 77
Colon, rectum, and anal canal, 143–166
  anal cancer, 165–166
  anal f ssure, 163
  anatomy, 144f
  anorectal abscess, 163
  anorectal f stula, 164
  anus, 145
  anus injury, 56
  benign tumors, 157, 158t
  blood supply, 145
  colitis, 148–151
  colon, 144f
  colorectal carcinoma (CRC), 158–160, 161t, 162f
  colorectal polyps, 157
  constipation, 147
  defecation, 147
  diarrhea, 148
  disorders of motility, 147–148
  diverticular disease, 151–153
  embryology, 144
  hemorrhoids, 161–163
  histology, 146
  inflammatory bowel disease, 150–151
  innervation, 146
  irritable bowel syndrome (IBS), 147
  large bowel obstruction, 154–155
  lower GI bleed (LGIB), 153, 154t
  lymphatic drainage, 146
  microbiology, 146
  Ogilvie syndrome, 156–157
  perianal and anal problems, 161–165
  physiology, 146–147
  pilonidal disease, 165

polyps, 157
pseudo-obstruction, 156–157
rectum, 144–145
rectum injury, 56
ulcerative colitis, 150–151, 151t
volvulus, 155, 156f
Colon cancer, 160
Colorectal carcinoma (CRC), 158–160, 161t, 162f
Colorectal polyps, 157
Colorectal resection, 160, 162f
Columns of Morgagni, 145
Comminuted fracture, 430–431
Commissurotomy, 386–387
Common bile duct (CBD), 201f, 217f
Common bile duct (CBD) obstruction, 207
Common bile duct (CBD) stones, 206–207
Compartment syndrome, 290, 434–435
Complete fasciotomy, 435
Complete SBO, 128
Complete spinal cord injuries, 375
Concussion, 365
Conductive hearing loss (CHL), 337
Congenital aganglionosis coli, 331
Congenital cystic adenomatoid malformation (CCAM), 324
Congenital deformities, 321–324
Congenital diaphragmatic hernia (CDH), 323
Congenital diseases of neck, 357–358
Conn's syndrome, 251
Constipation, 147
Contaminated wound, 27
Continuous positive airway pressure (CPAP), 71t
Cooper's hernia, 184
Cooper's ligaments, 266
Cooper's repair, 180
Corner stitch, 14
Coronary artery bypass grafting (CABG), 383–385
Coronary atherosclerosis, 382
Coronary ligament, 186f, 187
Corticospinal tract (CST), 376, 376f
Cortisol, 232t
Coumadin, 24
Courvoisier's sign, 209, 223
CPAP. See Continuous positive airway pressure (CPAP)
CPP. See Cerebral perfusion pressure (CPP)
Cranial foramina, 362
Craniopharyngioma, 372–373
Crawford classification of thoracoabdominal aneurysms, 398
CRC. See Colorectal carcinoma (CRC)
Cremasteric reflex, 418
Cricopharyngeus muscle, 92
Critical care, 61–72
ARDS, 71–72
mechanical ventilation, 70, 71t
pressors and inotropes, 68–70
shock, 62–66
Swan-Ganz catheter, 66–67
CRM. See Circumferential radial margin (CRM)
Crohn's disease, 131–133, 151t
Crossclamping of aorta, 52
Crossmatching, 403
Cruveilhier-Baumgarten bruit, 200
Cryostat, 432
Cryptorchidism, 414
Crystalloid, 77
CSF. See Cerebrospinal fluid (CSF)
CSF rhinorrhea, 365
CST. See Corticospinal tract (CST)
Cullen's sign, 51, 219
Curling's ulcer, 113
Current body water (CBW), 79
Cushing reflex, 364
Cushing's syndrome, 247, 248–249, 249t, 372
Cushing's ulcer, 113
CXR. See Chest radiograph (CXR)
Cyclosporine, 404
Cystadenocarcinoma, 224
Cystadenoma, 224
Cystectomy, 427
Cystic duct, 201f
Cystic hygroma, 321
Cystosarcoma phyllodes, 273
Cytokine release syndrome, 405
Cytomegalovirus (CMV) ulcer, 105

D

DAI. See Diffuse axonal injury (DAI)
Danger/alar space, 356
Darto's fascia, 414
DCIS. See Ductal carcinoma in situ (DCIS)
De Musset's sign, 390
De Quervain's thyroiditis, 240
DeBakey classification of thoracic aneurysm, 395
Debridement and skin grafting, 288
Deep cervical fascia, 355
Deep incisional SSIs, 29
Deep vein thrombosis (DVT), 22–23, 315–316
Defecation, 147
Degenerative aneurysm, 311
Dehiscence, 32–33
Dehydration, 75f, 76–77
Dehydroepiandrosterone (DHEA), 233t
Denonvilliers' fascia, 145
Dentate line, 145
Denys-Drash syndrome, 333
DES. See Diffuse esophageal spasm (DES)
Descending aortic aneurysm, 395
Descending arch aneurysm, 398
Descending colon, 144f
Desmopressin (DDAVP), 21
Dexamethasone, 371
DHEA. See Dehydroepiandrosterone (DHEA)
Diabetes insipidus, 256
Diabetic disease, 314
Diabetic neuropathy, 314
Diagnostic peritoneal lavage (DPL), 45, 53–54, 56
Dialysis, 21
Diaphragm, 186f
Diaphragmatic hernia, 323
Diarrhea, 148
Diesel fuel injector injuries, 458
Dieulafoy's lesion, 121
Diffuse axonal injury (DAI), 365
Diffuse esophageal spasm (DES), 94–95, 95t
Diffuse intracranial lesions, 365
Digital subtraction angiography (DSA), 304
Direct inguinal hernia, 181
Dirty/infected wound, 27
Dissecting aneurysm, 311
Distal splenorenal shunt, 198, 198f
Distraction or seat-belt injury, 380
Distributive shock, 63, 66t
Diverticular disease, 151–153
Diverticulectomy, 96
Diverticulitis, 152, 153
Diverticulopexy, 96
Diverticulosis, 152
Dobutamine, 68
Donut sign, 330
Dopamine, 69
Dopamine agonists, 68t
Double bubble sign, 329
DPL. See Diagnostic peritoneal lavage (DPL)
DSA. See Digital subtraction angiography (DSA)
Duct of Santorini, 216
Duct of Wirsung, 216
Ductal carcinoma in situ (DCIS), 275t
Ductal decompression/drainage procedures, 222
Ducts of Luschka, 200
Dumping syndrome, 114
Duodenal atresia, 328, 329
Duodenal blood supply, 125
Duodenal hematoma, 58
Duodenal perforation, 112
Duodenal ulcer, 109–111
Duodenojejunostomy, 216
Duodenum, 124–125
Duodenum injury, 56
Duroziez's sign, 390

Duval procedure, 222
DVT. See Deep vein thrombosis (DVT)
Dysphagia, 93

E

Ear, 336–340. See also Ear, nose, and throat surgery
  acoustic neuroma, 339–340
  air conduction/bone conduction, 337
  anatomy, 336–337
  cholesteatoma, 339
  conductive hearing loss (CHL), 337
  functional testing, 337–338
  otitis externa, 339
  otitis media, 338
  otitis media with effusion (OME), 338–339
  physiology, 337
  sensorineural hearing loss (SNHL), 337
  vestibular schwannoma, 339
Ear, nose, and throat surgery, 335–360. See also individual subentries
  ear, 336–340
  facial nerve, 340–341
  larynx and hypopharynx, 347–351
  neck, 354–360
  nose, 341–344
  oral cavity and oropharynx, 344–347
  salivary glands, 351–354
Eaton-Lambert syndrome, 393t
ECF. See Extracellular fluid (ECF)
ECG. See Electrocardiogram (ECG)
Echocardiography, 19, 383
Ectopic ACTH, 249
EGD. See Esophagogastroduodenoscopy (EGD)
Electrical alternans, 48f
Electrical injury, 289–290
Electrocardiogram (ECG)
  electrical alternans, 48f
  hypokalemia, 84f
  ischemic heart disease, 383
  tachycardia, 66
Electrolytes. See Fluids, electrolytes, and nutrition
Embolism, 305
Emergency dialysis, 87
Emphysematous cholecystitis, 204–205, 205f
Enchondroma, 440–441
End-to-side shunt, 198, 198f
Endocrine hormones, 230–233t
Endocrine pancreas, 217
Endocrine system, 229–256
  adenoma, 254–255
  adrenal gland. See Adrenal gland
  diabetes insipidus, 256
  hormones, 230–233t
  medullary tumors, 252–254
  multiple endocrine neoplasia (MEN), 233–234
  neuroblastoma, 252–253
  parathyroid. See Parathyroid gland
  pheochromocytoma, 253–254
  pituitary gland, 254–256
  Sheehan's syndrome, 255
  SIADH, 256
  thyroid. See Thyroid gland
Endocrine tumors, 224–227
Endoscope sclerotherapy, 97
Endoscopic dilatation, 94
Endoscopic retrograde cholangiopancreatography (ERCP), 188, 223
Endoscopic sclerotherapy, 97
Endoscopic ultrasound (EUS), 103
Endovascular repair (EVAR), 313, 398
ENT. See Ear, nose, and throat surgery
Entamoeba histolytica, 192
Enteral nutrition, 87, 88–89
Enteroclysis, 134
Ependymoma, 371
Epididymis, 415
Epidural anesthesia, 298, 299t
Epidural hematoma, 366, 366f
Epinephrine, 69, 233t, 300
Epistaxis, 343
Epithelialization, 27
Epstein-Barr virus (EBV), 344, 406
EPV. See Epstein-Barr virus (EBV)
ERCP. See Endoscopic retrograde cholangiopancreatography (ERCP)
ERCP with sphincterotomy, 188
Escharotomy, 287, 287f
Escherichia coli O157:H7, 258
Esophageal anastomoses, 92
Esophageal carcinoma, 102–105
Esophageal diverticula, 95–96
Esophageal hiatal hernia, 183–184
Esophageal manometry studies, 95, 100
Esophageal motility study, 93
Esophageal motor disorders, 93–96
Esophageal perforation/rupture, 97–98
Esophageal ulcers, 105
Esophageal varices, 96–97, 199–200
Esophageal web, 105
Esophagogastroduodenoscopy (EGD), 98, 103, 109, 200
Esophagomyotomy, 94
Esophagoscopy, 93, 95, 99, 100
Esophagus, 91–105
  achalasia, 93–94, 95t
  anatomy and physiology, 92
  assessment tools, 93
  Barrett's, 102
  caustic injury, 98–99
  diffuse esophageal spasm (DES), 94–95, 95t
  esophageal carcinoma, 102–105
  esophageal diverticula, 95–96
  esophageal motor disorders, 93–96
  esophageal perforation/rupture, 97–98
  esophageal ulcers, 105
  esophageal varices, 96–97
  GERD, 99–102
  nutcracker, 95
  Plummer-Vinson syndrome, 105
  Schatzki's ring, 105
  Zenker's diverticulum, 96
Essay award programs. See Medical student essay award
Essential thrombocythemia, 260t
Essentials *of* General Surgery (Lawrence), 10
ESWL. See Extracorporeal shockwave lithotripsy (ESWL)
EUS. See Endoscopic ultrasound (EUS)
EVAR. See Endovascular repair (EVAR)
Eventration, 184
Ewing's sarcoma, 436, 442–443
Excess scar formation, 33
Exocrine pancreas, 217
Exploratory laparotomy, 54
Extended right hemicolectomy, 162f
Extended small bowel enteroscopy, 134
Extensor zones of hand, 450f
External hernia, 176
Extracellular fluid (ECF), 74, 75f
Extracorporeal shockwave lithotripsy (ESWL), 422

F

Facial nerve, 340–341
Facial nerve paralysis, 340–341
FACTOID, 439
Falciform ligament, 186f, 187
False aneurysm, 311
Familial adenomatous polyposis (FAP), 134, 135t
Familial polyposis coli, 158t
FAP. See Familial adenomatous polyposis (FAP)
Fascial dehiscence, 31
Fasciotomy, 288, 435
FAST. See Focused abdominal sonogram for trauma (FAST)
Fat embolism syndrome, 431–432
Fat necrosis, 270–271
FAT RN, 258
Fatigue (stress) fracture, 430
FE_{NA}, 21
Fellowships. See Awards and opportunities
Felon, 451–452, 451f
Femoral artery aneurysm, 314
Femoral hernia, 182
Ferrous sulfate, 105
FESS. See Functional endoscopic sinus surgery (FESS)
FEV_1, 19
Fibroadenoma, 271–272

Fibrocystic changes, 272
Fibroma, 135t
Fick equation, 88
Fight for Sight summer student fellowship, 474
Fine needle aspiration (FNA), 241, 277
Finey strictureplasty, 133f
Fingers. See Hand
Fistula
  anorectal, 164
  H-type, 322, 323
  small bowel, 138
  TE, 322, 323f
Fitz-Hugh-Curtis syndrome, 41
FK-506, 404
Flank ecchymoses, 51
Flexible endoscopy, 93
Flexor tendon injury, 448, 449
Flexor zone II ("no man's land"), 449f, 451
Flexor zones of hand, 449f
Fluids, electrolytes, and nutrition, 73–90
  acid-based disorders, 89–90, 89t
  anion gap metabolic acidosis, 88, 89
  assessing volume status, 79–80
  body fluids, 74, 75f
  calcium balance, 83t, 85–87
  calories requirements, 88
  dehydration, 75f, 76–77
  emergency dialysis, 87
  enteral nutrition, 87, 88–89
  fluid and electrolyte exchange, 74, 75f, 75t
  free water def cit (FWD), 79
  gastrointestinal secretions, 78t
  HARD UP, 89
  hypercalcemia, 87
  hyperkalemia, 84–85
  hypernatremia, 81–83, 82t
  hypocalcemia, 86–87
  hypokalemia, 83–84
  hyponatremia, 81–82, 83t
  magnesium imbalance, 83t
  metabolic acidosis, 89–90, 89t
  metabolic alkalosis, 89t, 90
  MIS[HAP]^{3}S, 90
  MUDPILES, 88
  nutrient requirements, 88
  nutritional support, 87
  ongoing fluid loss, 79
  plasma osmolality, 74
  potassium balance, 83–85, 83t
  protein requirement, 88
  renal control of fluids/electrolytes, 76, 76f
  respiratory acidosis, 89t, 90
  respiratory alkalosis, 89t, 90
  respiratory quotient (RQ), 88
  signs/symptoms of volume disturbances, 75t
  sodium balance, 80–83
  total parenteral nutrition (TPN), 87
  volume def cit (dehydration), 75f, 76–77
  volume excess, 75f, 78–79
5-fluorouracil (5-FU), 160
FNA. See Fine needle aspiration (FNA)
FNH. See Focal nodular hyperplasia (FNH)
Focal intracranial lesions, 366–367
Focal nodular hyperplasia (FNH), 194
Focused abdominal sonogram for trauma (FAST), 45, 51–52, 52f, 53f
Follicle-stimulating hormone (FSH), 230t
Follicular thyroid cancer, 242–243t
Foramen cecum, 234
Fracture, 430–431
Free water def cit (FWD), 79
Frey procedure, 222
Frey's syndrome, 353
FRIENDS, 139
FSH. See Follicle-stimulating hormone (FSH)
Functional endoscopic sinus surgery (FESS), 342
Fundoplication procedure, 101–102, 101f
"Funnel chest" appearance, 321
FWD. See Free water def cit (FWD)

G

G. Milton Shy award, 467–468
Galeazzi fracture, 459t
Gallbladder, 186f, 201f
Gallbladder anatomy, 200
Gallbladder carcinoma, 209–210
Gallstone ileus, 208–209
Gallstones, 201–202
Galveston formula, 285
GAME PAD, 111
Gamekeeper's thumb, 453, 453f
Ganglion cyst, 455
Gardner's syndrome, 134, 158t
Garengoff's hernia, 184
Gastric adenocarcinoma, 117–120
Gastric adenocarcinoma TNM classif cation, 119–120t
Gastric lavage, 116
Gastric lymphoma, 120
Gastric outlet obstruction, 115
Gastric ulcer, 111–113
Gastric volvulus, 122
Gastrin, 127t
Gastrinoma, 225–226
Gastrinoma triangle, 225f
Gastritis, 113–114
Gastroesophageal reflux disease (GERD), 99–102
Gastrointestinal defects, 326–333
Gastrointestinal embryology, 124
Gastrointestinal hemorrhage, 115–116
Gastrointestinal hormones, 127t
Gastrointestinal secretions, 78t
Gastrointestinal stromal tumor (GIST), 120
Gastroschisis, 125, 324–325
Gaucher's disease, 261t
GCS. See Glasgow coma scale (GCS)
Genitourinary (GU) system, 413–428
  acute scrotum, 417–418
  anatomy, 414–416
  Bell-clap deformity, 414
  benign prostatic hyperplasia, 422–423
  hematuria, 416–417
  hydrocele, 417
  kidney stones, 420–422
  priapism, 417
  prostate cancer, 423–425
  renal cell carcinoma (RCC), 425–426
  seminoma, 419, 420
  testicular cancer, 419–420
  testicular torsion, 418–419
  transitional cell carcinoma of collecting system/ureters, 426
  urolithiasis, 420–422
  urothelial cancer, 426
Genitourinary (GU) trauma, 57–59
GERD. See Gastroesophageal reflux disease (GERD)
Gerota's fascia, 415
GH. See Growth hormone (GH)
Giant cell tumor, 441
Gigantism, 255
GIST. See Gastrointestinal stromal tumor (GIST)
Glasgow coma scale (GCS), 364, 365f
Gleason grading system for prostate cancer, 424
Glial tumor, 371
Glisson's capsule, 187
Glomus jugulare tumor, 373
Glomus tumors, 373
Glottis, 347
Glucagon, 127t, 232t
Glucagonoma, 226–227
Glucocorticoids, 250
GNASHING, 113
Golden hour of trauma, 44
Goldman's index (top risks), 18
Goldman's risk assessment for noncardiac surgery, 19f
Goodsall's rule, 164, 164f
Graft-versus-host disease (GVHD), 410
Gram-negative organisms, 63
Gram-positive organisms, 63
Granulation, 26
Granulomatosis with polyangiitis, 340–341
Grave's disease, 237–238
Grease gun injuries, 458
Greenstick fracture, 431
Grey-Turner's sign, 51, 219

Groin anatomy, 176–178, 177f
Groin hernia, 176, 179t
Groin hernia surgery, 180–181
Growth hormone (GH), 230t
Growth hormone-producing tumor, 255
GU system. See Genitourinary (GU) system
GU trauma. See Genitourinary (GU) trauma, 57–59
Gut, 126
GVHD. See Graft-versus-host disease (GVHD)
Gynecomastia, 273, 274

H

H pylori, 110
H-type f stula, 322, 323
Halothane hepatitis, 298
Hamartoma, 135t
Hamman's sign, 98
Hand, 445–449
  amputation injuries, 457
  Bennett fracture, 458t
  bones, 446–447, 448f
  boxer's fracture, 458t
  carpal tunnel syndrome, 454–455, 454f
  clinical maneuvers to test muscles, 448t
  Colles' fracture, 459t
  felon, 451–452, 451f
  Galeazzi fracture, 459t
  gamekeeper's thumb, 453, 453f
  ganglion cyst, 455
  high-pressure injection injuries, 458
  history, 450
  infections, 451–453
  mallet f nger, 455–456, 456f
  Monteggia fracture, 459t
  muscles, 446, 446t, 447t
  nerve blocks, 451
  nerves, 447
  nightstick fracture, 459t
  "no man's land," 449f, 451
  paronychia, 452, 452f
  physical examination, 450–451
  Rolando fracture, 458t
  "safe" position, 451f
  scaphoid fracture, 458t
  septic flexor tenosynovitis, 452–453
  Smith fracture, 459t
  STEP, 452
  tendons, 449, 449f, 450f
  trigger f nger, 456–457, 456f
  zones, 449, 449f, 450f
Hangman's fracture, 378, 379f
HARD UP, 89
Harris-Benedict equation, 88
Hartmann's procedure, 153
Hartman's pouch, 200
Hashimoto's thyroiditis, 240
Havard-Longwood summer research fellowship in vascular surgery, 469
Head trauma, 363–365, 367, 368
"Headlight" appearance (cholelithiasis), 202f
Heart block, 391
Heart transplant, 410–411
Heineke-Mikulicz strictureplasty, 132f
Helicobacter pylori, 110
Heller's myotomy, 94
Hemangioblastoma, 373
Hemangioma, 135t
Hemartoma, 193
Hematoma, 32
Hematuria, 416–417
Hemicolectomy, 162f
Hemodynamic parameters, 67t
Hemolytic uremic syndrome (HUS), 258
Hemorrhoidal complex, 145
Hemorrhoids, 161–163
Hemothorax, 49–50
Hepatic abscess, 191–192
Hepatic anatomy, 186–187, 186f
Hepatic cyst, 192–193
Hepatic encephalopathy, 198–199
Hepatic metastasis, 195–196, 196f
Hepatobiliary iminodiacetic acid (HIDA) scan, 187–188, 203, 203f, 204f
Hepatobiliary system, 185–213
  acalculous cholecystitis, 205–206
  acute cholangitis, 207
  ascending cholangitis, 207
  ascites, 199
  benign liver tumors, 193–194
  benign tumors of bile ducts, 210
  bile, 188–189
  biliary system, 200, 201f
  biliary tree anatomy, 200, 201f
  caput medusa, 200
  carcinoma of gallbladder, 209–210
  cavernous hemangioma, 193
  CBD stones, 206–207
  Child-Pugh score, 196t
  cholangiocarcinoma, 211–213
  cholangitis, 207
  cholecystitis, 202–206
  choledochal cyst, 210–211, 211f
  choledocholithiasis, 206–207
  cholelithiasis, 201–202
  conditions of gallbladder and biliary tree, 201–213
  diagnostic imaging, 187–188
  emphysematous cholecystitis, 204–205, 205f
  esophageal varices, 199–200
  focal nodular hyperplasia (FNH), 194
  gallbladder anatomy, 200
  gallstone ileus, 208–209
  gallstones, 201–202
  hemartoma, 193
  hepatic abscess, 191–192
  hepatic anatomy, 186–187, 186f
  hepatic cyst, 192–193
  hepatic encephalopathy, 198–199
  hepatocellular adenoma, 193–194
  hepatocellular carcinoma, 194–195
  hydatid cyst, 192
  jaundice, 189, 189t
  liver blood supply, 187
  liver failure, 196–197
  liver injury, 189–191
  malignant liver tumors, 194–196
  MELD score, 197
  metastatic neoplasms, 195–196
  nontraumatic liver conditions, 191–196
  portal hypertension, 197–198
  sclerosing cholangitis, 207–208, 208f
  SINGE, 207
Hepatocellular adenoma, 193–194
Hepatocellular carcinoma, 194–195
Hepatoduodenal ligament, 187
Hepatoma, 194
Hereditary elliptocytosis, 260t
Hereditary nonpolyposis colon cancer syndrome (HNPCC), 158t
Hereditary spherocytosis, 258, 260t
Hernia and abdominal wall problems, 175–184
  acquired umbilical hernia, 182–183
  anatomical triangles, 177–178
  children, 323, 326
  classif cation of hernias, 176
  diaphragmatic hernia, 323
  differential diagnosis, 179t
  esophageal hiatal hernia, 183–184
  femoral canal structures, 177
  femoral hernia, 182
  groin anatomy, 176–178, 177f
  groin hernia, 176, 179t
  groin hernia surgery, 180–181
  hernia, def ned, 176
  inguinal hernia, 178–179, 181–182, 326
  internal structures, 176
  layers of abdominal wall, 176
  nerves, 177
  other hernias, 184
  overview, 176
  pediatric umbilical hernia, 183
  pediatric wall defects. See Pediatric surgery
Herpes simplex virus (HSV) ulcer, 105
Hesselbach's triangle, 177
Heterotopic pancreas, 216
Heterotropic, 402
Heyde syndrome, 153
Hiatal hernia, 183–184
HIDA scan. See Hepatobiliary iminodiacetic acid (HIDA) scan
Hidradenitis suppurativa, 270
High output f stula, 138
High-pressure injection hand injuries, 458
High radical inguinal orchiectomy, 420

Highly selective vagotomy, 111, 111f
Hilar cholangiocarcinoma, 212f
Hill's sign, 390
Hinchey staging system, 153
Hirschsprung's disease, 331–332
HIVED, 368
HNPCC. See Hereditary nonpolyposis colon cancer syndrome (HNPCC)
Hodgkin's disease, 260t
Horizontal mattress suture, 13
Hormones, 230–233t
Horner syndrome, 393t
Hospital for Special Surgery summer research fellowship, 475
House-Brackmann scale, 341
HSV ulcer. See Herpes simplex virus (HSV) ulcer
HUS. See Hemolytic uremic syndrome (HUS)
Hydatid cyst, 192
Hydrocele, 178, 417
Hydrocephalus, 368–370
Hydronephrosis, 415
Hydrops of gallbladder, 202
Hyoid bone, 347
Hyperaldosteronism, 251
Hypercalcemia, 87
Hypercalcemic crisis, 244
Hyperkalemia, 84–85
Hypernatremia, 81–83, 82t
Hyperparathyroidism, 244–245
Hyperplasia, 247
Hyperthyroidism, 237–239
Hypertrophic scar, 33
Hypoaldosteronism, 252
Hypocalcemia, 86–87, 246
Hypokalemia, 83–84
Hyponatremia, 81–82, 83t
Hyponatremic hypervolemia, 81
Hypoparathyroidism, 245–246
Hypopharynx, 347. See also Larynx and hypopharynx
Hypotension, 364
Hypothyroidism, 239
Hypovolemic shock, 62–63, 66t

I

IABP. See Intra-aortic balloon pump (IABP)
IBS. See Irritable bowel syndrome (IBS)
IBW. See Ideal body weight (IBW)
ICF. See Intracellular fluid (ICF)
ICP. See Intracranial pressure (ICP)
Ideal body weight (IBW), 21
Idiopathic facial nerve paralysis (Bell's palsy), 340
Idiopathic thrombocytopenic purpura, 260t
IgA. See Immunoglobulin A (IgA)
Ileocecal valve, 144f, 145
Ileocecectomy, 162f
Ileum, 125
Ileus, 22
Iliac artery aneurysm, 314
IMA, Inferior mesenteric artery (IMA)
Immunoglobulin A (IgA), 126
Imperforate anus, 331
IMV. See Inferior mesenteric vein (IMV)
Incarcerated hernia, 176
Incisional hernia, 32–33, 184
Incomplete spinal cord injuries, 375
Increased ICP, 368
Incus, 336, 336f
Indirect inguinal hernia, 176, 181–182, 326
Inferior mesenteric artery (IMA), 145
Inferior mesenteric vein (IMV), 145
Inferior rectal vein, 145
Inferior shoulder dislocation, 433, 433t, 434
Inferior vena cava, 186f
Inf ltrating ductal carcinoma, 274
Inf ltrating lobular carcinoma, 274
Inflammatory bowel disease, 150–151
Infrainguinal disease, 307
Infundibulum, 200
Inguinal canal, 176
Inguinal hernia, 178–179, 181–182, 326
Inguinal incision, 415
Innervation of stomach, 108
INR, 20
Insulin, 24, 232t
Insulin pump, 408
Insulinoma, 224
Intercostobrachial nerve, 268t
Intermittent bowel obstruction, 134
Internal hernia, 176
Internal iliac artery, 145
Internal iliac vein, 145
Internal pudendal artery, 145
Interpectoral nodes, 267f
Interstitial fluid, 75f
Interval appendectomy, 171
Intestinal atresia, 325, 328–329
Intra-aortic balloon pump (IABP), 65
Intracellular fluid (ICF), 74, 75f
Intracerebral hemorrhage, 366
Intracranial pressure (ICP), 364, 368
Intraductal papilloma, 273
Intraglandular lymphatics, 234
Intraoperative embolectomy, 140
Intraparietal hernia, 176
Intravenous pyelography (IVP), 427
Intussusception, 329–330
Inverting papilloma, 344
Irreducible hernia, 176
Irritable bowel syndrome (IBS), 147
Ischemia, 306–309
Ischemic colitis, 149–150
Ischemic heart disease, 382–383
Ischemic or strangulated bowel, 38t
Islet cell tumors, 224–227
Islet of Langerhans, 217f
Islet of Langerhans transplant, 408
Isograft, 402
Isoperistaltic side-to-side strictureplasty, 133f
Isoproterenol, 68–69
IVP. See Intravenous pyelography (IVP)

J

Jaundice, 189, 189t
Jefferson fracture, 377, 378f
Jejunoileal atresia, 329
Jejunum, 125, 126
JNA. See Juvenile nasopharyngeal angiof broma (JNA)
Juvenile nasopharyngeal angiof broma (JNA), 344

K

Kanavel signs of tenosynovitis, 452
Karmody poster competition, 463
Kasabach-Merritt syndrome, 193
Kehr's sign, 36, 51
Keloids, 33
Kidney, 415, 416f
Kidney stones, 420–422
Kidney transplant, 406–408
Kiesselbach's plexus, 343
Klatskin tumor, 212
Kocher's incision, 206
Korey award, 468
Krukenberg's tumor, 118
Kugel's method, 180

L

La-Fraumeni syndrome, 276
Laparoscopic appendectomy technique, 172f
Laparoscopy, 54
Large bowel obstruction, 154–155
Laryngeal cancer, 350–351
Laryngeal foreign bodies, 349
Laryngeal papilloma, 350
Laryngeal polyps, 349
Laryngectomy, 350
Laryngotracheal separation, 47
Larynx and hypopharynx, 347–351
  anatomy, 347, 347f, 348f
  benign lesions, 349–350
  evaluation of larynx, 349
  foreign bodies, 349
  functions of larynx, 348
  laryngeal cancer, 350–351
  lymphatic drainage, 348
  nerve supply, 348
  papilloma, 350
  physiology, 348–349
  polyps, 349
  singer's nodules, 349
  tracheotomy vs. laryngectomy, 350

Lateral pectoral nerve, 267, 268f
LCIS. See Lobular carcinoma in situ (LCIS)
Left lower quadrant (LLQ) pain, 39
Left-sided colon cancer, 145, 160
Left upper quadrant (LUQ) pain, 39
Left ventricular (LV) function, 387
Leiomyoma, 135t
Leiomyosarcoma, 136t
Leriche syndrome, 306
LES. See Lower esophageal sphincter (LES)
Levator ani, 145
LeVeen shunt, 199
LGIB. See Lower GI bleed (LGIB)
LH. See Luteinizing hormone (LH)
Lichtenstein's repair, 180
Lidocaine, 342
Ligament of Treitz, 125
Ligamentum teres, 186f
LILT. See Longitudinal intestinal lengthening and tailoring (LILT)
Lingual thyroid, 237
Lipoma, 135t
Littre's hernia, 184, 326
Liver. See Hepatobiliary system
Liver blood supply, 187
Liver failure, 196–197
Liver injury, 189–191. See Hepatobiliary system
Liver transplant, 198, 409–410
LLQ pain. See Left lower quadrant (LLQ) pain
Lobular carcinoma in situ (LCIS), 275t
Local anesthesia, 299
Long thoracic nerve (of Bell), 268t
Longitudinal intestinal lengthening and tailoring (LILT), 142
Low back pain, 437–438, 439
Lower esophageal sphincter (LES), 92
Lower GI bleed (LGIB), 153, 154t
Ludwig's angina, 358–359
Lumbar hernia, 184
Lumpectomy and axillary node dissection, 278
Lung cancer, 391–395
Lung transplant, 411–412
LUQ pain. See Left upper quadrant (LUQ) pain
Luteinizing hormone (LH), 231t
LV function. See Left ventricular (LV) function
Lymphatics, 234
Lymphoma, 136t
Lynch syndrome, 158t

M

MacEwen's sign, 368
Mackay award, 465–466
Macroadenoma, 254, 255
Macrophages, 26
Mafenide acetate, 286t
Magnesium-ammonium-phosphate stones, 421
Magnesium imbalance, 83t
Magnetic resonance angiography (MRA), 303, 308
Major histocompatibility complexes (MHCs), 402–403
Malignant hyperthermia, 299–300
Mallampati classif cation of ease of intubation, 18, 18f
Mallet f nger, 455–456, 456f
Mallory-Weiss syndrome, 97, 98, 115
Malrotation and midgut volvulus, 328
MALT. See Mucosa-associated lymphoid tissue (MALT)
Mammary duct ectasia, 272
Mammography, 277, 277f
Manometry, 93
MAP. See Mean arterial blood pressure (MAP)
Marjolin's ulcer, 291
Mastectomy, 268, 278
Mastitis, 270
Maydl's hernia, 184
McBurney's point, 38, 169
McVay's repair, 180
Mean arterial blood pressure (MAP), 364
Meatus, 341
Mechanical ventilation, 70, 71t
Meckel's diverticulum, 124, 330–331
Medial pectoral nerve, 267, 268f
Medical student essay award
  extended neuroscience award, 466–467
  Korey award, 468
  Mackay award, 465–466
  Samuels essay award competition, 462–463
  Shy award, 467–468
Medullary endocrine tumors, 252–254
Medullary thyroid cancer, 242–243t
MEDVIPS, 218
Meissner plexus, 125
MELD score. See Model for end-stage-liver disease (MELD) score
MEN. See Multiple endocrine neoplasia (MEN)
MEN I, 225, 233
Men IIA, 233
MEN IIB, 234
Ménétrier's disease, 121
Meningioma, 372
Mesenteric ischemia, 139–141
Mesh repair, 180
Mesoappendix, 168
Mesocaval H graft, 198, 198f
Metabolic acidosis, 89–90, 89t
Metabolic alkalosis, 89t, 90
Metastatic bone tumors, 444
Metastatic lymphadenopathy, 359–360
Metyrapone test, 249t
MHCs. See Major histocompatibility complexes (MHCs)
MI. See Myocardial infarction (MI)
Michael S. Pessin stroke leadership prize, 466
Microadenoma, 254, 255
Microcytic anemia, 160
Microhematuria, 416
MIDCAB. See Minimally invasive direct CABG (MIDCAB)
Middle rectal vein, 145
Midgut volvulus, 130f, 328
Milan criteria, 195
Milrinone, 69
Minimally invasive direct CABG (MIDCAB), 383
Minute ventilation, 70
Mirizzi's syndrome, 202
Mirror examination, 349
MIS[HAP]^{3}S, 90
Mitral insuff ciency, 387–388
Mitral stenosis, 385–387
Mixed alpha and beta agonists, 68t
MMF. See Mycophenolate mofetil (MMF)
Model for end-stage-liver disease (MELD) score, 19t, 197, 409
Modif ed radical neck dissection, 359
Mondor's disease, 272
Monomorphic adenoma, 353
Monro-Kellie hypothesis, 364
Monteggia fracture, 459t
Morrison's pouch, 52
Motilin, 127t
Motility disorders, 92
MRA. See Magnetic resonance angiography (MRA)
Mucinous appendiceal tumors, 174
Mucoepidermoid carcinoma, 354
Mucosa-associated lymphoid tissue (MALT), 126
MUDPILES, 88
Multiple endocrine neoplasia (MEN), 233–234
Multiple hereditary osteochondroma, 440
Multiple myeloma, 443–444
Mumps, 353
Mycophenolate mofetil (MMF), 404
Mycotic aneurysm, 311
Myelof brosis, 260t
Myocardial infarction (MI), 65

N

Nasal endoscopy, 342
Nasal lymphoma, 344
Nasal masses, 343–344
Nasal melanoma, 344
Nasal polyps, 343
Natural orif ce transluminal endoscopic surgery (NOTES), 174
NAVEL, 303

NBME examination, 9
NBW. See Normal body water (NBW)
NEC. See Necrotizing enterocolitis (NEC)
Neck, 354–360
  abscess, 358
  anatomy, 354–356
  branchial apparatus anomalies, 357–358
  branchial arches, 357t
  children (masses/lesions), 320–321
  congenital diseases, 357–358
  fascial layers of retrovisceral space, 356f
  fascial planes, 355f
  infectious/inflammatory lesions, 358–359
  Ludwig's angina, 358–359
  lymphatic drainage, 357
  lymphatic malformation, 358
  lymphoma, 359
  malignancies, 359–360
  metastatic lymphadenopathy, 359–360
  neck dissection, 359
  nerve supply, 356
  penetrating neck injuries, 46–47
  retropharyngeal abscess, 359
  thyroglossal duct cyst, 358
  thyroid/parathyroid tumors. See Endocrine system
  triangles, 355f
  vascular supply, 356
  zones, 46, 47f
Neck abscess, 358
Neck dissection, 359
Neck trauma, 46–47
Necrolytic migratory erythema, 226
Necrotizing enterocolitis (NEC), 325, 332–333
Neoplastic disease, 333–334
Nephrectomy, 426
Nephrolithiasis, 422f
Nephroureterectomy, 426
Neuroblastoma, 252–253, 334
Neurogenic shock, 65, 66t, 375
Neuroma, 372
Neuromuscular blockade, 296–297
Neurosurgery, 361–380
  astrocytoma, 371
  brain tumors, 370–374
  carotid body tumor, 373
  cerebral concussion, 365
  cerebral contusion, 366
  circle of Willis, 363, 363f
  CNS tumors, 370–371
  CNS vasculature, 362
  cranial foramina, 362
  craniopharyngioma, 372–373
  diffuse axonal injury (DAI), 365
  diffuse intracranial lesions, 365
  ependymoma, 371
  epidural hematoma, 366, 366f
  focal intracranial lesions, 366–367
  Glasgow coma scale (GCS), 364, 365f
  glial tumor, 371
  glomus tumors, 373
  head trauma, 363–365, 367, 368
  hemangioblastoma, 373
  HIVED, 368
  hydrocephalus, 368–370
  increased ICP, 368
  intracerebral hemorrhage, 366
  low back pain, 437–438, 439
  meningioma, 372
  metastatic tumors, 373–374
  neuroma, 372
  oligodendroglioma, 371
  pituitary adenoma, 372
  SCALP, 363
  severe head injury, 368–370
  skull fracture, 365
  spinal trauma, 374–380. See also Spinal trauma
  spondylosis, etc., 374
  subdural hematoma, 366–367, 367f
  tuberous sclerosis clinical triad, 374
  Von Hippel-Lindau disease, 373
Neurotensin, 127t
Neurovascular bundle, 49
NH_3, 20
Niemann-Pick disease, 261t
Nightstick fracture, 459t
Nipple discharge, 273–274
Nitrogen balance, 88
Nitrous oxide, 298
"No man's land," 449f, 451
Non-Hodgkin's lymphoma, 260t
Non-small cell lung cancer, 392, 393t, 394
Non-ST elevation MI (NSTEMI), 384
Nonanion gap metabolic acidosis, 89
Nonsteroidal antiinflammatory drugs (NSAIDs), 24
Norepinephrine, 69, 233t
Normal body water (NBW), 79
Nose, 341–344. See also Ear, nose, and throat surgery
  acute bacterial rhinosinusitis, 342
  anatomy, 341
  blood supply, 341
  carcinoma, 344
  chronic sinusitis, 342
  epistaxis, 343
  evaluation of nasal cavity, 342
  FESS, 342
  inverting papilloma, 344
  juvenile nasopharyngeal angiofibroma (JNA), 344
  lymphoma, 344
  malignancies, 344
  melanoma, 344
  nasal masses, 343–344
  nerve supply, 341
  physiology, 342
  polyps, 343
  sinus disease, 342
Nosebleed, 343
NOTES. See Natural orifice transluminal endoscopic surgery (NOTES)
NPO, 23
NSAIDs. See Nonsteroidal antiinflammatory drugs (NSAIDs)
NSTEMI. See Non-ST elevation MI (NSTEMI)
Nuss procedure, 321
Nutcracker esophagus, 95
Nutrient requirements, 88
Nutritional deficiencies, 114
Nutritional support, 87
NYHA classification of heart disease, 383
NYU Hospital for Joint Diseases Department of Orthopaedics year-long fellowship, 470
NYU urology summer fellowship, 470–471

O

Objective structured clinical examination (OSCE), 9
Obstipation, 147
Obstructive shock, 66, 66t
Odontoid fracture, 377, 378f
Odynophagia, 93
Ogilvie syndrome, 156–157
OKT3, 404–405
Oligodendroglioma, 371
Ollier's disease, 440
OME. See Otitis media with effusion (OME)
Omphalocele, 125, 325–326
Oncocytoma, 353
Open appendectomy, 171f
Open commissurotomy, 386–387
Open fracture, 430
Operative case log, 3t
Operative note, 8t
Oral cavity and oropharynx, 344–347
  anatomy, 344–345
  blood supply, 345
  malignancies, 346–347
  nerve supply, 345
  oral cavity carcinoma, 346
  oropharyngeal carcinoma, 346–347
  peritonsillar abscess, 345
  physiology, 345
  ranula, 346
  tonsillectomy, 345
Oral cavity carcinoma, 346
Orchiectomy, 420
Oregon Health and Science University–Campagna scholarship in neurological surgery, 462
Organ of Zuckerkandl, 253
Organ/space SSIs, 30
Organ transplantation. See Transplant
Oropharyngeal carcinoma, 346–347
Oropharynx, 345

Orthopedic Research and Education Foundation fellowship, 472–473
Orthopedics, 429–444
bone tumors, 438–444
Brodie's abscess, 437
chondrosarcoma, 442
compartment syndrome, 434–435
enchondroma, 440–441
Ewing's sarcoma, 442–443
FACTOID, 439
fat embolism syndrome, 431–432
fracture, 430–431
giant cell tumor, 441
low back pain, 437–438, 439
metastatic bone tumors, 444
multiple myeloma, 443–444
Ollier's disease, 440
osteoblastoma, 439–440
osteochondroma, 440
osteoid osteoma, 439
osteomyelitis, 435–437
osteosarcoma, 441–442
PAM, 443
POEMS, 443
septic bursitis, 437
shoulder dislocation, 433–434, 433t
Orthotropic, 402
OSCE. See Objective structured clinical examination (OSCE)
Osler-Weber-Rendu disease, 343
Osmolar gap, 74
Osmotic diuresis, 79
Ossicles, 336
Osteitis f brosa cystica, 245
Osteoblastoma, 439–440
Osteochondroma, 440
Osteoid osteoma, 439
Osteomyelitis, 435–437
Osteoporosis, 430
Osteosarcoma, 441–442
Otitis externa, 339
Otitis media, 338
Otitis media with effusion (OME), 338–339
Oxytocin, 231t

P

Paget's disease, 165, 274
PAM, 443
Pancoast's syndrome, 393t
Pancreas, 201f, 215–227
acute pancreatitis, 218–220
adenocarcinoma, 222–223
anatomy, 216, 217f
blood supply and innervation, 216
chronic pancreatitis, 220–222
cystadenocarcinoma, 224
cystadenoma, 224
embryology, 216
endocrine tumors, 224–227
gastrinoma, 225–226
glucagonoma, 226–227
insulinoma, 224
islet cell tumors, 224–227
MEDVIPS, 218
physiology, 217
Ransom's criteria, 219t
somatostatinoma, 227
transplantation, 408–409
trauma. See Trauma
VIPoma, 226
Pancreas divisum, 216
Pancreas transplant, 408–409
Pancreatic adenocarcinoma, 222–223
Pancreatic cystadenoma, 224
Pancreatic injury, 57
Pancreatic polypeptide, 127t, 232t
Pancreatitis, 41, 218–222
Panel reactive antibodies (PRA), 403
Pantaloon hernia, 184
PAOP. See Pulmonary artery occlusion pressure (PAOP)
Papillary thyroid cancer, 242–243t
Papillotomy, 188
Paraesophageal hiatal hernia, 183, 184
Paralytic ileus, 38t, 126
Paranasal sinuses, 341
Paraneoplastic syndromes, 393
Parathyroid cancer, 246
Parathyroid gland, 243–246
anatomy, 243
calcitonin, 244
development, 243
hyperparathyroidism, 244–245
hypoparathyroidism, 245–246
parathyroid cancer, 246
physiology/calcium homeostasis, 243
PTH, 243–244
Parathyroid hormone (PTH), 231t, 243–244
Paravertebral plexus of Batson, 267
Parietal cell vagotomy, 111
Parinaud's syndrome, 369, 371
Parkland formula, 285
Paronychia, 452, 452f
Partial SBO, 128
Patent processus vaginalis, 178
Pathologic fracture, 430
Patient experience. See Surgical patient
Patients' rights, 4
Patterson-Kelly syndrome, 105
Pco_2, 71
PCWP. See Pulmonary capillary wedge pressure (PCWP)
PE. See Pulmonary embolism (PE)
Peau d'orange, 268
Pectus carinatum, 321–322
Pectus excavatum, 321
Pediatric airway, 298, 298t
Pediatric surgery, 319–334
abdominal wall defects, 324–326
biliary atresia, 327–328
bilious vomiting, 328
branchial cleft cyst, 320–321
congenital aganglionosis coli, 331
congenital cystic adenomatoid malformation (CCAM), 324
congenital deformities, 321–324
congenital diaphragmatic hernia (CDH), 323
cystic hygroma, 321
fluid management, 320
gastrointestinal defects, 326–333
gastroschisis, 324–325
Hirschsprung's disease, 331–332
imperforate anus, 331
inguinal hernia, 326
intestinal atresia, 328–329
intussusception, 329–330
malrotation and midgut volvulus, 328
Meckel's diverticulum, 330–331
neck masses/lesions, 320–321
necrotizing enterocolitis (NEC), 332–333
neoplastic disease, 333–334
neuroblastoma, 334
omphalocele, 325–326
pectus carinatum, 321–322
pectus excavatum, 321
pulmonary sequestration, 323–324
pyloric stenosis, 326–327
tracheoesophageal (TE) f stula, 322, 323f
tracheoesophageal (TE) malformations, 322–323
VACTERL anomalies, 322, 331
Wilm's tumor, 333–334
Pediatric umbilical hernia, 183
PEEP. See Positive end-expiratory pressure (PEEP)
Penetrating neck injuries, 46–47
Penis, 414, 414f
Peptic ulcer disease (PUD), 109, 111
Peptide YY, 127t
Percutaneous balloon valvuloplasty, 387
Percutaneous nephrolithotomy, 422
Percutaneous transhepatic cholangiography (PTCA), 188, 223
Percutaneous transluminal angioplasty (PTA), 307
Percutaneous transluminal coronary angioplasty (PTCA), 384–385
Perforated viscus, 38t
Pericardial tamponade, 48
Perineal hernia, 184
Peripheral vascular disease (PVR), 305
Peristalsis, 92, 126
Peritoneal irritation, 169
Peritonitis, 38t
Peritonsillar abscess, 345
Periumbilical ecchymosis, 51
Pessin stroke leadership prize, 466

Peter B. Samuels essay award competition, 462–463
Petrosal sinus sampling, 249t
Peutz-Jeghers syndrome, 134, 135t, 158t
Phalen's test, 454f
Phenylephrine, 70
Pheochromocytoma, 253–254
Phyllodes tumor, 273
"Pigeon chest" appearance, 322
Pilonidal disease, 165
Pituitary adenoma, 372
Pituitary anatomy, 254, 254f
Pituitary gland, 254–256
Plain film, 393
Plasma, 75f
Plasma cell mastitis, 272
Plasma osmolality, 74
Plasmapheresis, 261
Platelet count, 21t
Platelet dysfunction, 298
Plavix, 24
Pleomorphic adenoma, 353
Plethysmography, 303
Plicae circulares, 125, 126
Plummer-Vinson syndrome, 105
Plummer's disease, 238–239
Plunging ranula, 346
Pneumonia, 20
Pneumothorax, 48–49, 49f
Po_2, 71
Pocket cards, 13–14
POEMS, 443
Poiseuille's law, 304
Polycystic kidney disease, 192
Polycythemia vera, 260t
Polyps, 157
Popliteal artery aneurysm, 314
Popliteal pulse, 310
Portacaval anastomoses, 198f
Portacaval H graft, 198
Portal hypertension, 197–198
Positive end-expiratory pressure (PEEP), 70
Posterior fossa mass, 371
Posterior pituitary disorders, 256
Posterior shoulder dislocation, 433, 433t, 434
Postgastrectomy complications, 114
Postoperative bleeding, 21t
Postoperative note, 8t
Postphlebitic syndrome, 310, 316
Poststenotic aneurysm, 311
Posttransplant lymphoproliferative disease (PTLD), 406
Postvagotomy diarrhea, 114, 148
Potassium balance, 83–85, 83t
Pouch of Douglas, 52
PQRST, 36
PRA. See Panel reactive antibodies (PRA)
Prehn's sign, 418
Preoperative note, 7t
Preparing patient for surgery. See Surgical patient
Presensitization (transplant), 405
Pressors and inotropes, 68–70
Pressure support ventilation (PSV), 71t
Priapism, 417
Primary intention, 28, 28f
Primary peristalsis, 92
Pringle maneuver, 191
PRL. See Prolactin (PRL)
Procedure note, 7t
Prolactin (PRL), 231t
Prolactinoma, 255, 372
Prostate, 415
Prostate cancer, 423–425
Prostate-specific antigen (PSA), 424
Prostatic hyperplasia, 422–423
Protein requirement, 88
Proximal fistula, 138
Proximal gastric vagotomy, 111
PSA. See Prostate-specific antigen (PSA)
Pseudo-kidney sign, 330
Pseudoaneurysm, 303
Pseudohyperkalemia, 84
Pseudomembranous colitis, 148
PSV. See Pressure support ventilation (PSV)
PTA. See Percutaneous transluminal angioplasty (PTA)
PTCA. See Percutaneous transhepatic cholangiography (PTCA); Percutaneous transluminal coronary angioplasty (PTCA)
PTH. See Parathyroid hormone (PTH)
PTLD. See Posttransplant lymphoproliferative disease (PTLD)
PUD. See Peptic ulcer disease (PUD)
Puestow procedure, 222
Pulmonary artery catheter, 66
Pulmonary artery occlusion pressure (PAOP), 67
Pulmonary capillary wedge pressure (PCWP), 65, 66t, 67
Pulmonary embolism (PE), 22–23, 23t
Pulmonary sequestration, 323–324
Pulse volume recording (PVR), 303
Pulsion diverticula, 96
PVR. See Peripheral vascular disease (PVR); Pulse volume recording (PVR)
Pyloric stenosis, 326–327
Pyruvate kinase deficiency, 260t

Q

Quadrantectomy and axillary radiotherapy (QUART), 278
QUART. See Quadrantectomy and axillary radiotherapy (QUART)
Quincke's sign, 390

R

Raccoon's eyes, 365
Radiation-induced colitis, 148–149, 149t
Radical cystectomy, 427
Radical mastectomy, 278
Radical neck dissection, 359
Radical nephrectomy, 426
Radioactive iodide uptake test (RAIU), 238
Radiofrequency ablation (RFA), 196
RAIU. See Radioactive iodide uptake test (RAIU)
Raloxifene, 281
Ramstedt pyloromyotomy, 327
Ransom's criteria, 219t
Ranula, 346
Rapid-sequence intubation, 296
RCC. See Renal cell carcinoma (RCC)
Rectal cancer, 160
Rectum, 144–145, 144f. See also Colon, rectum, and anal canal
Rectum injury, 56
Reducible hernia, 176
Reference materials, 10
Renal cell carcinoma (RCC), 425–426
Renal injury, 58, 59f, 59t
Renin, 251
Research opportunities. See Awards and opportunities
Respiratory acidosis, 89t, 90
Respiratory alkalosis, 89t, 90
Respiratory quotient (RQ), 88
Reteplase, 305
Retrograde cystogram, 58
Retrograde urethrogram, 58
Retroperitoneal lymph node dissection (RPLND), 420
Retroperitoneal x-ray therapy, 420
Retropharyngeal abscess, 359
Retrovisceral space, 356f
RFA. See Radiofrequency ablation (RFA)
Rhabdomyolysis, 59
Rheumatic fever, 436
Rheumatoid arthritis, 375f
Richter's hernia, 184
Riedel's fibrosing thyroiditis, 240
Right hemicolectomy, 162f
Right lower quadrant (RLQ) pain, 39, 131
Right recurrent laryngeal nerve (RLN), 236
Right-sided colon cancer, 145, 160
Right upper quadrant (RUQ) pain, 38–39
Rinne test, 337–338
RLN. See Right recurrent laryngeal nerve (RLN)
RLQ pain. See Right lower quadrant (RLQ) pain
Rolando fracture, 458t

Rosewell Park Cancer Institute summer oncology research program, 463
Ross procedure, 389
Rotter's nodes, 267, 267f
Roux-en-Y gastric bypass, 116, 117f
RPLND. See Retroperitoneal lymph node dissection (RPLND)
RQ. See Respiratory quotient (RQ)
Rule of 1/3s for abdominal stab, 50
Rule of nines, 284, 285f
Rumbling diastolic murmur, 391
Running sutures, 13
Ruptured diaphragm, 55f
Ruptured esophageal varices, 200
Ruptured omphalocele, 325
RUQ pain. See Right upper quadrant (RUQ) pain

S

Sacral sparing, 375
"Safe" position of hand, 451f
Salivary ducts, 344
Salivary gland tumors, 353
Salivary glands, 351–354
  acinic cell carcinoma, 354
  adenocarcinoma, 354
  adenoid cystic carcinoma, 354
  anatomy, 351
  benign/systemic disease, 352–353
  benign tumors, 353
  carcinoma ex pleomorphic, 354
  Frey's syndrome, 353
  malignant tumors, 354
  monomorphic adenoma, 353
  mucoepidermoid carcinoma, 354
  mumps, 353
  oncocytoma, 353
  physiology, 351–352
  pleomorphic adenoma, 353
  radiographic evaluation, 352
  sialoadenitis, 352–353
  sialolithiasis, 352
  Sjögren's syndrome, 352
  tumors, 353
  Warthin's tumor, 353
Salter-Harris fracture, 430–431, 431f
Samuels essay award competition, 462–463
Sarcoma, 136t
Saul R. Korey award, 468
SBO. See Small bowel obstruction (SBO)
SCALP, 363
Scaphoid fracture, 458t
Scar formation, 33
Schatzki's ring, 105
Scholarships. See Awards and opportunities
Schwartz's Principles *of* General Surgery, 10
Sclerosing adenosis, 273
Sclerosing cholangitis, 207–208, 208f
"Scoring" of pulses, 303
Scrotum, 414
Seat-belt injury, 380
Seat-belt sign, 50
Secondary intention, 28–29, 28f
Secondary peristalsis, 92
Secretin, 127t, 217
Segmental blood pressure measurement, 303
Selective neck dissection, 359
Seminoma, 419, 420
Sengstaken-Blakemore tube, 97, 200
Sensorineural hearing loss (SNHL), 337
Sentinel loop, 220
Sentinel node biopsy, 280
Sepsis, 64t
Septic arthritis, 436
Septic bursitis, 437
Septic flexor tenosynovitis, 452–453
Septic insult, 64f
Septic shock, 64t
Serial hematocrits, 54
Serial transverse enteroplasty procedure (STEP), 142
Seroma, 32
Severe head injury, 368–370
Severe sepsis, 64t
Sheehan's syndrome, 255
"Shelf" examination, 9
Shock, 62–66
  ABCs, 62
  anaphylactic, 63–65
  cardiogenic, 65–66, 66t
  defined, 62
  distributive, 63, 66t
  hypovolemic, 62–63, 66t
  medications, 68–70
  neurogenic, 65, 66t
  obstructive, 66, 66t
  pathophysiology, 62
  septic, 64t
  spinal, 65
  tissue perfusion, 62
Short bowel syndrome, 141–142
Shoulder dislocation, 433–434, 433t
Shouldice repair, 180
Shy award, 467–468
SIADH. See Syndrome of inappropriate antidiuretic hormone (SIADH)
Sialoadenitis, 352–353
Sialolithiasis, 352
Sickle cell disease, 260t
Side-to-side shunt, 198, 198f
Sigmoid colectomy, 162f
Sigmoid colon, 144f
Sigmoid volvulus, 155, 156f
SIGN AAN neurology program, 464
Silk glove sign, 326
Silver nitrate, 286t
Silver sulfadiazine, 286t
Simple interrupted suture, 13
SIMV. See Synchronized intermittent mandatory ventilation (SIMV)
SINGE, 207
Singer's nodules, 349
Sinus disease, 342
Sinusitis, 342
Sipple's syndrome, 233
SIRS. See Systemic inflammatory response syndrome (SIRS)
Sister Mary Joseph's nodule, 118
Sistrunk's operation, 237
Sjögren's syndrome, 352
Skin grafting, 288
Skull fracture, 365
Sliding esophageal hernia, 183, 184
Sliding inguinal hernia, 184
SMA. See Superior mesenteric artery (SMA)
Small bowel, 123–142
  absorption, 128t
  benign neoplasms, 134, 135t
  carcinoid syndrome, 137–138
  Crohn's disease, 131–133
  duodenal blood supply, 125
  duodenum, 124–125
  fistula, 138
  FRIENDS, 139
  GI embryology, 124
  GI hormones, 127t
  gross anatomy, 124–126
  ileum, 125
  innervation, 125
  jejunum, 125, 126
  lymphatics, 125
  malignant neoplasms, 134–137
  mesenteric ischemia, 139–141
  peristalsis, 126
  short bowel syndrome, 141–142
  small bowel obstruction (SBO), 126–131
  superior mesenteric artery (SMA), 126, 139
Small bowel obstruction (SBO), 126–131
Small bowel transplant, 410
Small bowel transplantation, 142
Small cell lung cancer, 391–392, 393t, 394
Small intestine, 124. See also Small bowel
Smith fracture, 459t
SMV. See Superior mesenteric vein (SMV)
SNHL. See Sensorineural hearing loss (SNHL)
"Soap bubble" appearance, 441
Society for Clinical and Vascular Surgery–Peter B. Samuels essay award competition, 462–463
Sodium balance, 80–83
Solitary thyroid nodule, 241f
Somatostatin, 127t, 218, 232t
Somatostatinoma, 227

Specif c gravity, 21
Spermatic cord, 176
SPF-10, 300
Sphincter of Oddi, 125, 216
Sphincterotomy, 211
Spigelian hernia, 184
Spinal anesthesia, 298, 299, 299t
Spinal cord, 376f
Spinal cord injuries, 375–376
Spinal cord syndromes, 376–377
Spinal shock, 65, 375
Spinal trauma, 374–380
  anatomy, 374
  anterior cord syndromes, 376
  Brown-Séquard syndrome, 376–377
  central cord syndrome, 377
  cervical spine f lms, 377
  cervical spine fracture, 377–379
  Chance fracture, 380
  clay shoveler's fracture, 379, 380f
  distraction or seat-belt injury, 380
  hangman's fracture, 378, 379f
  Jefferson fracture, 377, 378f
  neurogenic shock, 375
  odontoid fracture, 377, 378f
  pathology and pathophysiology, 374
  seat-belt injury, 380
  spinal cord, 376f
  spinal cord injuries, 375–376
  spinal cord syndromes, 376–377
  spinal shock, 375
  thoracic outlet syndrome (TOS), 380
  thoracic spine fracture, 379
  thoracolumbar junction and lumbar spine fracture, 379–380
Spinothalamic tract (STT), 376, 376f
Spiral computed tomography (CT), 304
Spiral valves of Heister, 200
Spleen, 257–263
  AAST spleen injury scale, 262t
  anatomic boundaries, 258, 259f
  FAT RN, 258
  splenectomy, 258, 260–261t
  splenic abscess, 262–263
  splenic injury, 261–262
  splenic rupture, 258, 262
  tumors, 258
Splenectomy, 258, 260–261t
Splenic abscess, 262–263
Splenic injury, 261–262
Splenic rupture, 258, 262
Splenomegaly, 198
Splenorenal recess, 52
Splenorenal shunt, 198, 198f
Split-thickness skin graft (STSG), 288
Spondylolisthesis, 374
Spondylolysis, 374
Spondylosis, 374
Spray gun injuries, 458
SSIs. See Surgical site infections (SSIs)
ST elevation MI (STEMI), 384
Stanford classif cation of thoracic aneurysm, 395
Stapes, 336, 336f
STEMI. See ST elevation MI (STEMI)
STEP, 452. See Serial transverse enteroplasty procedure (STEP)
Steroids, 403–404
Stertor, 349
Stomach, 107–122
  adenocarcinoma, 117–120
  adenomatous polyps, 121
  afferent loop syndrome, 114
  alkaline reflux gastritis, 114
  anatomy, 108f
  bariatric surgery, 116, 117f
  benign lesions, 121–122
  blood supply, 108
  Curling's ulcer, 113
  Cushing's ulcer, 113
  Dieulafoy's lesion, 121
  dumping syndrome, 114
  duodenal ulcer, 109–111
  GAME PAD, 111
  gastric lymphoma, 120
  gastric outlet obstruction, 115
  gastric ulcer, 111–113
  gastric volvulus, 122
  gastritis, 113–114
  gastrointestinal hemorrhage, 115–116
  gastrointestinal stromal tumor (GIST), 120
  GNASHING, 113
  histology and physiology, 108–109
  innervation, 108
  malignant tumors, 117–120
  Ménétrier's disease, 121
  nutritional def ciencies, 114
  peptic ulcer disease (PUD), 109, 111
  postgastrectomy complications, 114
  postvagotomy diarrhea, 114
  trauma, 55
Stomach injury, 55
Stoppa's repair, 180
Straight leg-raising test, 438
Strangulated bowel, 38t, 129
Strangulated hernia, 176
Streptokinase, 305
Stress fracture, 430
Stress test, 18
Strictureplasty, 132f, 133f
Stridor, 349
Struvite stones, 421
STSG. See Split-thickness skin graft (STSG)
STT. See Spinothalamic tract (STT)
Student experience. See Surgery clerkship
Subdural hematoma, 366–367, 367f
Subglottis, 347
Subtotal colectomy, 162f
Succinylcholine, 296
Superf cial cervical fascia, 354
Superf cial femoral vein, 315
Superf cial incisional SSIs, 29
Superf cial venous thrombosis (SVT), 316
Superior mesenteric artery (SMA), 126, 139, 145
Superior mesenteric vein (SMV), 145
Superior vena cava syndrome, 393t
Supraglottis, 347
Supratentorial mass, 371
Surgery clerkship, 1–14
  awards. See Awards and opportunities
  bullet presentation, 5
  chest radiograph (CXR), 5–7
  clerkship grade, 8–9
  clinical clerkship examination, 11–12
  dress/attire, 3
  honesty, 4
  how to study?, 9–11
  keep patient information handy, 5
  notes (procedure, operative, etc.), 7t, 8t
  operating room, 2
  patients' rights, 4
  pocket cards, 13–14
  reference materials, 10
  research opportunities. See Awards and opportunities
  respectfulness, 4
  team player, 4
  timeliness, 3
  wards, 3–8
Surgical antimicrobial prophylaxis, 31
Surgical pathogens, 30
Surgical patient, 17–24
  antibiotic prophylaxis, 21
  ASA classif cation, 296t
  bowel preparation, 24
  C di*ff* colitis, 22
  cardiac risk assessment, 18
  common complications, 22–23
  deep vein thrombosis (DVT)/pulmonary embolism (PE), 22–23, 23t
  general postoperative complications, 21–22
  Goldman's index (top risks), 18
  Goldman's risk assessment for noncardiac surgery, 19f
  hematological assessment, 20–21
  hepatic risk assessment, 19–20
  ileus, 22
  instructions to patient, 23–24
  medication, temporary discontinuance, 24
  MELD score, 19t
  NPO, 23
  nutritional assessment, 21
  preoperative evaluation, 18
  pulmonary risk assessment, 18–19
  renal risk assessment, 20
  risk of postoperative bleeding, 21t
  wound infection. See Wound healing
Surgical site infections (SSIs), 29–30, 32
Surgical wound classif cation, 27
Suture technique, 13–14
SVR. See Systemic vascular resistance (SVR)

SVRI. See Systemic vascular resistance index (SVRI)
SVT. See Superf cial venous thrombosis (SVT)
Swan-Ganz catheter, 66–67
"Swiss cheese" appearance, 323
Syme's amputation, 309
Synchronized intermittent mandatory ventilation (SIMV), 71t
Syndrome of inappropriate antidiuretic hormone (SIADH), 256, 393t
Systemic inflammatory response syndrome (SIRS), 64t
Systemic vascular resistance (SVR), 62, 67
Systemic vascular resistance index (SVRI), 67

T

T_3, 231t
T_4, 231t
Tachycardia
  ECG f ndings, 66
  hypovolemic shock, 63
Tacrolimus, 404
Tamoxifen, 280–281
TAPP. See Transabdominal preperitoneal procedure (TAPP)
Target sign, 330
TBNA. See Transbronchial needle aspiration (TBNA)
TBW. See Total body water (TBW)
TE f stula. See Tracheoesophageal (TE) f stula
TE malformations. See Tracheoesophageal (TE) malformations
TEE. See Transesophageal echocardiogram (TEE)
TENS. See Transcutaneous electrical nerve stimulation (TENS)
Tension pneumothorax, 49
TEP. See Totally extraperitoneal procedure (TEP)
Tertiary intention, 28f, 29
Testes, 414–415
Testicular cancer, 419–420
Testicular torsion, 417, 418–419
TFTs. See Thyroid function tests (TFTs)
TH. See Thyroid hormone (TH)
Thalassemia, 260t
Thenar atrophy, 454
Thermodilutional technique, 67
Third-space losses, 79
Third spacing, 78
Thoracentesis, 394
Thoracic aortic aneurysm, 395–398
Thoracic outlet syndrome (TOS), 380
Thoracic spine fracture, 379
Thoracic trauma, 48–50
Thoracoabdominal aneurysm, 398–399
Thoracodorsal nerve, 268t
Thoracolumbar junction and lumbar spine fracture, 379–380
Thoracotomy, 50
Throat. See Ear, nose, and throat surgery
Thrombolytic therapy, 306
Thrombosis, 305–306
Thrombotic thrombocytopenic purpura, 261t
Thyroglossal duct cyst, 234, 237, 320, 358
Thyroid blood supply, 236f
Thyroid cancer, 242–243t
Thyroid function tests (TFTs), 238
Thyroid gland, 234–243
  anatomy, 234, 235f, 236
  assessment of function, 237
  congenital anomalies, 237
  development, 234
  Grave's disease, 237–238
  hormones, 236–237
  hyperthyroidism, 237–239
  hypothyroidism, 239
  innervation, 236
  Plummer's disease, 238–239
  solitary thyroid nodule, 241f
  thyroid blood supply, 236f
  thyroid cancer, 242–243t
  thyroid storm, 239
  thyroiditis, 240
  thyrotoxicosis, 239
  toxic nodular goiter, 238–239
  workup of a mass, 240–241
Thyroid hormone (TH), 236–237
Thyroid-stimulating hormone (TSH), 230t
Thyroid storm, 239
Thyroiditis, 240
Thyrotoxicosis, 239
Thyroxine, 24
Tinel's test, 455
TIPS. See Transjugular intrahepatic portacaval shunt (TIPS)
TIPSS. See Transjugular intrahepatic portosystemic shunt (TIPSS)
Tissue perfusion, 62
Tissue plasminogen activator (tPA), 305
Tissue typing, 403
TME. See Total mesorectal excision (TME)
TNB. See Transthoracic needle biopsy (TNB)
TNM classif cation system of colorectal cancer, 160
TNM staging for breast cancer, 279t
Todani classif cation of choledochal cysts, 210, 211f
Toe amputation, 309
Tonsillectomy, 345
Topical decongestants, 342
TOS. See Thoracic outlet syndrome (TOS)
Total body water (TBW), 74
Total colectomy, 162f
Total mesorectal excision (TME), 160
Total parenteral nutrition (TPN), 87
total proctocolectomy, 162f
Totally extraperitoneal procedure (TEP), 180, 180f
Toxic nodular goiter, 238–239
tPA. See Tissue plasminogen activator (tPA)
TPN. See Total parenteral nutrition (TPN)
Tracheoesophageal (TE) f stula, 322, 323f
Tracheoesophageal (TE) malformations, 322–323
Tracheostomy, 47, 350
Tracheotomy, 350
Transabdominal preperitoneal procedure (TAPP), 180, 180f
Transbronchial needle aspiration (TBNA), 394
Transcutaneous electrical nerve stimulation (TENS), 438
Transesophageal echocardiogram (TEE), 50, 397
Transillumination, 417
Transitional cell carcinoma of collecting system/ureters, 426
Transjugular intrahepatic portacaval shunt (TIPS), 200
Transjugular intrahepatic portosystemic shunt (TIPSS), 97
Transmetatarsal amputation, 309
Transplant, 401–412
  brain death, 402
  cardiac, 410–411, 412
  crossmatch, 403
  def nitions, 402
  immunology, 402–403
  immunosuppression, 403–405
  kidney, 406–408
  liver, 409–410
  lung, 411–412
  major histocompatibility complexes (MHCs), 402–403
  organ preservation, 406
  pancreas, 408–409
  presensitization, 405
  rejection, 405–406
  small bowel, 410
  tissue typing, 403
Transplant immunology, 402–403
Transplant rejection, 405–406
Transrectal ultrasonography (TRUS), 424
Transthoracic echocardiography (TTE), 397
Transthoracic needle biopsy (TNB), 394
Transurethral prostatectomy (TURP), 423
Transurethral resection of bladder tumor (TURBT), 427

Transverse colectomy, 162f
Transverse colon, 144f
Trauma, 43–59
  ABCs, 44–45
  abdominal, 50–56
  advanced trauma life support (ATLS), 44–45
  AMPLE history, 46
  bladder rupture, 58
  blunt cardiac, 48
  genitourinary (GU), 57–59
  golden hour, 44
  head. See Neurosurgery
  hemothorax, 49–50
  neck, 46–47
  pancreatic injury, 57
  pericardial tamponade, 48
  pneumothorax, 48–49, 49f
  radiologic and diagnostic studies, 45
  renal injury, 58, 59f, 59t
  resuscitation, 45–46
  spinal. See Neurosurgery
  tension pneumothorax, 49
  tetanus prophylaxis, 46
  thoracic, 48–50
  ureteral injury, 58
Trauma laparotomy, 57
Trauma resuscitation, 45–46
Traumatic aneurysm, 311
Triangle of Grynfeltt, 177
Triangle of Petit, 178
Triangular ligaments, 186f, 187
Tricuspid stenosis, 391
Trigger f nger, 456–457, 456f
Trousseau's syndrome, 393t
True aneurysm, 311
True vocal cords, 347
Truncal vagotomy, 148
TRUS. See Transrectal ultrasonography (TRUS)
TSH. See Thyroid-stimulating hormone (TSH)
TTE. See Transthoracic echocardiography (TTE)
Tuberous sclerosis clinical triad, 374
Turbinates, 341
TURBT. See Transurethral resection of bladder tumor (TURBT)
Turcot's syndrome, 158t
TURP. See Transurethral prostatectomy (TURP)
Tympanic membrane, 336
Type I hypersensitivity reaction, 65

U

U_{NA}, 21
UA. See Unstable angina (UA)
UES. See Upper esophageal sphincter (UES)
Ulcerative colitis, 150–151, 151t
Umbilical hernia, 182–183
University of California at Davis ophthalmology summer fellowship, 468–469
University of Texas M.D. Anderson Cancer Center summer research program in biomedical sciences, 463–464
University of Wisconsin–Madison surgery summer research experience, 473–474
Unna boots, 311
Unstable angina (UA), 384
Upper esophageal sphincter (UES), 92
Upper extremity amputation, 310
Upper extremity disease, 307–308
Upper GI hemorrhage, 115–116
Urease breath test, 110
Ureter, 415
Ureteral injury, 58
Ureteroscopy, 422
Urethra, 416
Uric acid stones, 421
Urinalysis, 58
Urokinase, 305
Urolithiasis, 420–422
Urologic injury scale, 59t
Urothelial cancer, 426

V

VACTERL anomalies, 322, 331
Valentine student research grant in urology, 462
Valsalva maneuver, 145, 177
Valve prostheses, 391
Valvular heart disease, 385–391
Varicocele, 415
Vas deferens, 415
Vascular insuff ciency, 302
Vascular physical exam, 302–303
Vascular surgery, 301–317
  abdominal aortic aneurysm (AAA), 312–314
  acute arterial occlusion, 304–306
  acute compartment syndrome (ACS), 316–317
  amputation, 309–310
  anatomy, 302
  aneurysm, 311–314
  aortoiliac disease, 306–307
  carotid artery stenosis, 308–309
  chronic ischemia, 306–309
  deep vein thrombosis (DVT), 315–316
  diabetic disease, 314
  diagnostic tests, 303–304
  embolism, 305
  infrainguinal disease, 307
  obstruction and incompetence, 310–311
  physical exam, 302–303
  postphlebitic syndrome, 316
  signs of vascular insuff ciency, 302
  superf cial venous thrombosis (SVT), 316
  thrombosis, 305–306
  upper extremity disease, 307–308
  vascular trauma, 316–317
  venous thrombotic disease, 314–316
Vascular trauma, 316–317
Vasoactive drugs, 68, 68t
Vasopressin, 231t
VBG. See Vertical banded gastroplasty (VBG)
Venous thrombotic disease, 314–316
Ventriculoperitoneal (VP) shunt, 369, 370f
Ventriculostomy, 368
Verner-Morrison syndrome, 226
Vertical banded gastroplasty (VBG), 116, 117f
Vertical mattress suture, 13
Vertigo, 340
Vestibular apparatus, 337
Vestibular schwannoma, 339
Videostroboscopy, 349
VIPoma, 226
Virchow's node, 118
Vitamin B_{12} def ciency, 108
Vitelline duct, 124
VO_2, 19
Vocal cords, 347, 349–350
Volume def cit (dehydration), 75f, 76–77
Volume excess, 75f, 78–79
Volume expansion, 77
Volvulus, 155, 156f
Von Hippel-Lindau disease, 373
VP shunt. See Ventriculoperitoneal (VP) shunt

W

WAGR syndrome, 333
Waldeyer's fascia, 145
Warfarin, 24
Warm-antibody autoimmune hemolytic anemia, 260t
Warren shunt, 198, 198f
Warthin's tumor, 353
WDHA syndrome, 226
Weber test, 337
Wedge pressure, 68
Wegener's granulomatosis, 340–341
Well's criteria for PE, 23
Wermer's syndrome, 233
Whipple procedure, 134, 222, 223
Whipple's triad, 224

Wide pulse pressure, 390
William J. von Leibig summer research fellowship in vascular surgery, 471–472
Wilm's tumor, 333–334
Wolff-Chaikoff effect, 238
Wound dehiscence, 32–33
Wound failure, 32–33
Wound healing, 25–33
  angiogenesis, 26
  antimicrobial prophylaxis, 31–32
  coagulation, 26
  collagen synthesis, 26
  contraction and remodeling, 27
  dehiscence, 32–33
  epithelialization, 27
  excess scar formation, 33
  factors to consider, 29t
  granulation, 26
  hematoma, 32
  incisional hernia, 32–33
  inflammation, 26
  primary intention, 28, 28f
  secondary intention, 28–29, 28f
  seroma, 32
  surgical pathogens, 30
  surgical site infections (SSIs), 29–30, 32
  surgical wound classif cation, 27
  tertiary intention, 28f, 29
  wound failure, 32–33
  wound infections, 29–30, 31
Wound infections, 29–30, 31
Wrist injuries, 458–459t. See also Hand
www.mdconsult.com, 10

X

Xenograft, 402

Y

Yersinia enterolytica, 170

Z

Zenker's diverticulum, 96
Zollinger-Ellison syndrome, 109, 110, 225
Zones of the hand, 449, 449f, 450f
Zones of the neck, 46, 47f